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American Heart Journal

VOL. 53 JUNE, 1957 NO. 6

## Original Communications

# EARLY AGE OF MENOPAUSE IN YOUNG WOMEN WITH FATAL ACUTE MYOCARDIAL INFARCTION

ROBERT S. SPITZER, M.D., KYU TAIK LEE, M.D., AND WILBUR A. THOMAS, M.D.

ST. LOUIS, MO.

IN AN effort to obtain further information concerning sex hormonal factors and acute myocardial infarction,[1-4] we have reviewed the autopsy records of 500 patients (334 men, 166 women) who died with acute myocardial infarction in Barnes Hospital (1910-1954). Our attention has been directed especially to the age of menopause in women as a reflection of the hormonal state, with particular emphasis on the age of menopause in young women who died from acute myocardial infarction. The purpose of this report is to present the results of this study.

### MATERIAL AND METHODS

The autopsy and clinical records of 500 patients with acute myocardial infarction from Barnes Hospital (myocardial infarction group—1910-1954) were reviewed. This group has been characterized in several ways in previous reports.[5,6]

The age of menopause was determined for all women for whom such information was available in the clinical history (80 of the 166 women with acute myocardial infarction). Sixteen of the 80 women were less than 53 years of age at the time of death and 64 women were 53 years or more.

To aid in evaluating the data obtained from the patients with acute myocardial infarction, similar data were obtained for comparison from an additional group of autopsied patients. Patients of similar age with diseases having no known relationship to the age at menopause were sought. Although a completely satisfactory group is not available in an autopsy series, the postoperative neurosurgical patients in our autopsy series seem to be reasonably satisfactory. The time between the onset of serious symptoms and death in most of these patients was short, as it frequently is in patients who die from myocardial infarction.

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This study was supported in part by Grant H-1820, from the National Heart Institute, Institute of Health, Public Health Service, Bethesda, Md.

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TABLE I. AGE OF ONSET OF MENOPAUSE AND OTHER DATA IN WOMEN WITH ACUTE MYOCARDIAL INFARCTION WHO DIED BEFORE 53 YEARS OF AGE COMPARED WITH A CONTROL GROUP

| MYOCARDIAL INFARCTION GROUP | | | | CONTROL GROUP* | | | |
|---|---|---|---|---|---|---|---|
| PATIENT NUMBER AND RACE | AGE AT DEATH | AGE AT MENOPAUSE | YEARS BETWEEN MENOPAUSE AND DEATH | PATIENT NUMBER | AGE AT DEATH | AGE AT MENOPAUSE | YEARS BETWEEN MENOPAUSE AND DEATH |
| 1 N | 35 | still menstruating | 0 | 1 | 37 | still menstruating | 0 |
| 2 W | 40 | 32 | 8 | 2 | 39 | still menstruating | 0 |
| 3 N | 40 | 39 | months | 3 | 41 | 40 | 1 |
| 4 W | 43 | 43 | months | 4 | 41 | still menstruating | 0 |
| 5 N | 43 | 43 | months | 5 | 44 | still menstruating | 0 |
| 6 W | 46 | still menstruating | 0 | 6 | 45 | 45 | months |
| 7 W | 47 | 38 | 9 | 7 | 46 | 46 | months |
| 8 N | 48 | 46 | 2 | 8 | 47 | still menstruating | 0 |
| 9 W | 48 | 46 | 2 | 9 | 49 | still menstruating | 0 |
| 10 W | 49 | 39 | 10 | 10 | 50 | 47 | 3 |
| 11 N | 49 | 47 | 2 | 11 | 51 | still menstruating | 0 |
| 12 W | 49 | 48 | 1 | 12 | 52 | 47 | 5 |
| 13 W | 50 | 43 | 7 | 13 | 52 | 50 | 2 |
| 14 W | 50 | 42 | 8 | | | | |
| 15 W | 52 | 41 | 9 | | | | |
| 16 W | 52 | 41 | 9 | | | | |
| Average | 46.4 | 14 of 16 | 4.2 (4.9 for 14 who reached menopause before death) | | 46.5 | 6 of 13 | 0.9 (2.0 for 6 who reached menopause before death) |

*Explained in the text. W—white, N—Negro.

There would seem to be no more reason why the house officer or medical student taking the medical history should be more alerted to the importance of the menstrual history in a patient with myocardial infarction than in a neurosurgical patient. Hence, neurosurgical patients more than 36 years of age who died within one month after a craniotomy for primary brain disease (mostly brain tumor) were chosen. Patients with a primary diagnosis of cerebral arteriosclerosis or pituitary adenoma were not included, but, otherwise, this group represented a consecutive series of all such patients operated on at Barnes Hospital from 1942 through 1954. This group will be referred to hereinafter as the "neurosurgical group." Thirteen of these 27 women were below 53 years of age and 14 were above 53 years.

## RESULTS

The results are summarized in Table I.

Sixteen of the women with acute myocardial infarction (young myocardial group) were less than 53 years of age at death. (According to most authorities, the usual age of menopause is 44 to 52.[7]) The average age at death of this group was 46.4 years. Fourteen of the 16 (87.5 per cent) had reached menopause. In the neurosurgical group, there were 13 patients who died between the ages of 37 and 53 (young neurosurgical group). The average age at death of this group was 46.5 years. Only 6 of the 13 (46.2 per cent) had reached menopause. Despite the small number in the samples, the difference between these percentages (87.5 per cent and 46.2 per cent) is statistically significant ($p = 0.02$ using the Chi-square test).

The significance test of 14/16 against 6/13 does not prove that the two groups are comparable, of course. Also it does not make adequate allowance for age, but any more elaborate attempt to correct for age would not be worth while because the data at best can give only a suggestion of a possible relationship.

In 7 patients in the young myocardial infarction group, intervals of 7 years or more had elapsed between menopause and death. Only 1 patient was found in the young neurosurgical group whose death occurred an appreciable time after her menopause (5 years). There were 5 Negro women out of the 16 in the young myocardial infarction group. Four of these 5 had undergone menopause before death.

In the myocardial infarction group of 16 women less than 53 years of age, a history of "hypertension" was found in 15, angina pectoris in 6, diabetes in 4, previous acute myocardial infarction in 1, uremia in 3, cancer of the breast with metastasis in 2, and cerebral hemorrhage in 1. Both patients (No. 1 and No. 6 in Table I) classified as not having reached the menopause at the time of death had complicated histories. Patient No. 1 had subacute glomerulonephritis, hypertensive heart disease, gangrene of the right leg, anemia, and uremia. Patient No. 6 had malignant hypertension and was being treated for "apresoline disease" when her terminal acute myocardial infarction occurred. (She had had a previous myocardial infarction 6 months before death.) This patient had one ovary surgically removed 4 years before death, and her menses when last described 1 year before death were scanty and irregular. So, it is possible that she had actually reached menopause.

The average age at which menopause had occurred among all women with acute myocardial infarction who died after 52 years of age (on whom such data were available) was 47.6 years. The corresponding age among the neurosurgical group was 47.4. The difference between the average ages for the two groups is not significant. Many of the women who died with acute myocardial infarction had myocardial scars indicating healed infarcts, but none had a medical history of infarction prior to their menopause.

## DISCUSSION

These data suggest that some factor associated with the menstrual cycle possibly protects women against acute myocardial infarction. Eighty-seven per cent of women who died with acute myocardial infarction at Barnes Hospital before the age of 53 years had reached menopause. On the other hand, only 46 per cent of the patients in the control group of corresponding age had reached the menopause.

The difference between these percentages (87 and 46) is statistically significant. However, it must be emphasized that we are dealing with small numbers; that the statistical test does not prove that the two groups are comparable; and that it is not possible to make adequate allowance for age. Hence, our data can only suggest a possible relationship.

It is interesting to note that many of the women in all age groups in this series with acute myocardial infarction had serious complicating diseases, such as hypertension and diabetes mellitus.[5,6] However, unless these complicating diseases result in an early menopause (and no evidence has been presented indicating that they affect the age of menopause) their presence would not influence the conclusions that have been drawn here.

## SUMMARY

The records of 500 autopsied patients who died with acute myocardial infarction have been reviewed and data pertaining to the possible relationship between sex hormonal factors and acute myocardial infarction have been presented.

Sixteen young women in this series died with acute myocardial infarction before the age of 53, and 14 of these had reached menopause. Only 6 of 13 women of nearly similar age in a control group had reached menopause. This difference (14 out of 16 compared with 6 out of 13) is statistically significant. Although the significance test does not establish the comparability of the two groups, it does appear that young women who die with myocardial infarction usually have had an early menopause. The conclusion seems warranted that some factor associated with the menstrual cycle possibly protects young women against acute myocardial infarction.

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# THE ELECTROCARDIOGRAM IN TETANUS

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SAN JUAN, PUERTO RICO.

MANY reports have appeared in the literature giving electrocardiographic findings encountered in different types of diseases of infectious origin.[1-9] Variable, nonspecific electrocardiographic alterations indicating myocardial damage have been described in some cases of diphtheria.[3-5] It is generally accepted that this damage is due to the effect of the diphtheria toxin upon the cardiac muscle.

In tetanus, as in diphtheria, a powerful toxin is produced which accounts for the clinical manifestations and severity of the disease.

In an attempt to determine whether the tetanus toxin produces a physiologic derangement on the heart by direct action upon the cardiac muscle and its extrinsic nerves or indirectly through its effect upon the medullary centers, this study has been undertaken.

## MATERIAL AND RESULTS

Electrocardiographic tracings including standard, augmented unipolar and precordial leads were obtained in 14 cases of generalized tetanus that were admitted to our service from October, 1955, to April, 1956. The age of the patients ranged from 8 to 63 years. Careful evaluation ruled out concomitant organic heart disease that could alter the electrocardiogram. The diagnosis was established according to the generally accepted criteria.[10,11]

Self-retaining electrodes, as described by Welsh,[12] were utilized. The limb electrodes were applied to the inguinal regions and shoulder areas, as advised by Pastor,[13] in order to eliminate interference from the muscular tremors characteristic of the disease. The tracings were obtained when there was no evidence of respiratory embarrassment or obstruction of the airways so as to avoid anoxia that could cause false electrocardiographic alterations. The electrocardiographic findings encountered in the 14 cases of generalized tetanus are presented in Table I.

As stated in Table I, sinus tachycardia occurred in six instances. This ranged from 104 to 130 beats per minute. Three of these patients had mild fever. Inversion of the T wave from $V_1$ to $V_3$ occurred in five cases. This con-

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stitutes the so-called juvenile pattern. It appeared in patients younger than 15 years of age.

Elevation of the S-T segment was found in 3 patients. These changes were fleeting. Fig. 1 illustrates the complete electrocardiogram obtained the day of admission on a 31-year-old patient with generalized tetanus who had been sick at home for 7 days. S-T elevation is noted in $V_4$. Follow-up electrocardiogram taken 3 months later failed to reveal any deviation of the S-T segment from the isoelectric line.

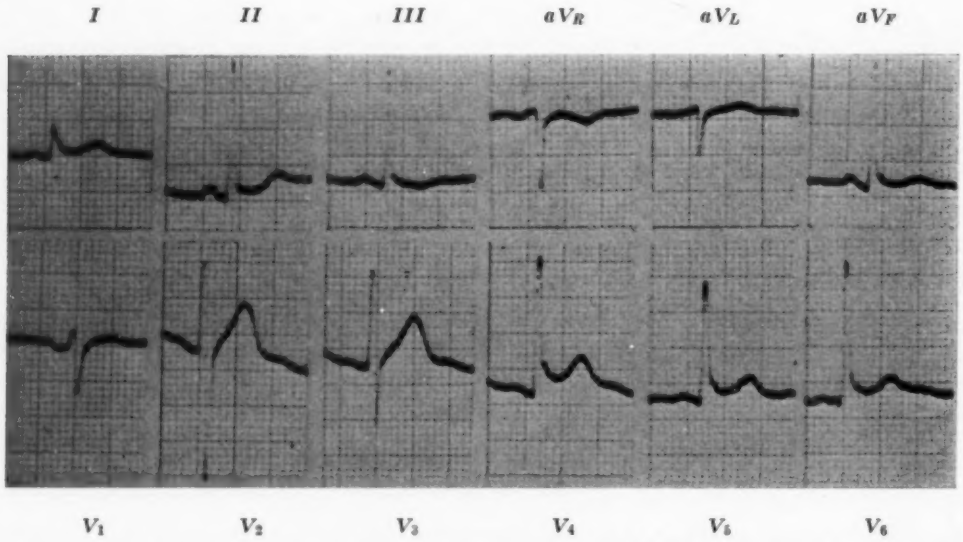


**Fig. 1.—Complete electrocardiogram taken the day of admission in a 31-year-old patient with tetanus.**

TABLE I. ELECTROCARDIOGRAPHIC FINDINGS IN 14 CASES OF TETANUS

| | |
|---|---|
| Sinus tachycardia | 6 |
| Prolonged Q-T interval | 5 |
| Juvenile pattern | 5 |
| S-T elevation | 3 |
| Premature ventricular beats | 2 |
| P-wave changes | 2 |
| Premature auricular beats | 1 |
| Nodal tachycardia | 1 |
| Sinus arrhythmia | 1 |
| Low QRS amplitude | 1 |

Fig. 2 shows the precordial leads taken in a 54-year-old patient with the disease. Mild initial elevation of the S-T segment is noted from $V_4$ to $V_6$ (Oct. 7, 1955) but it eventually disappeared (Oct. 24, 1955). Fleeting P-wave changes from $V_1$ to $V_6$ can also be noted in that tracing.

Prolongation of the Q-T interval was encountered in five cases. The upper limits of normal were determined according to the calculations in Ashman and Hull tables.[14]

Low QRS voltage occurred in one instance. Fig. 3, *A* illustrates the standard leads obtained in that patient. There was no associated condition in this patient known to produce electrical insulation and decrease in voltage.

Nodal tachycardia occurred once in a 61-year-old patient with the disease. It is illustrated by the standard leads shown in Fig. 3, *B*.

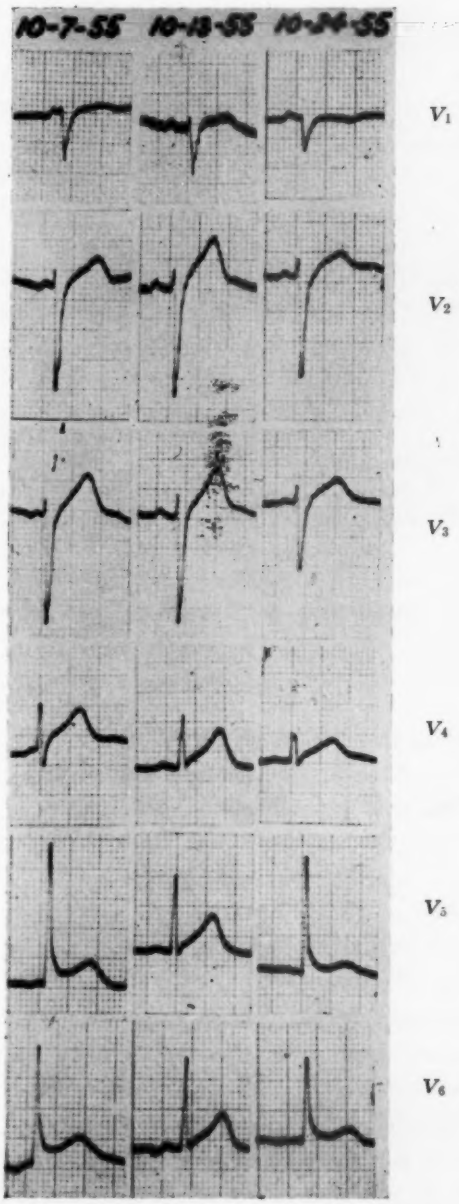


Fig. 2.—Serial precordial electrocardiographic tracings in a 64-year-old patient with tetanus.

Transitory P-wave changes were found in 2 patients. The tracings in Fig. 3, *C* illustrate such a finding in one of our patients. The precordial leads shown in Fig. 2 also illustrate fleeting P-wave changes.

Ectopic beats were also found. Two patients had premature ventricular beats while one had premature auricular beats.

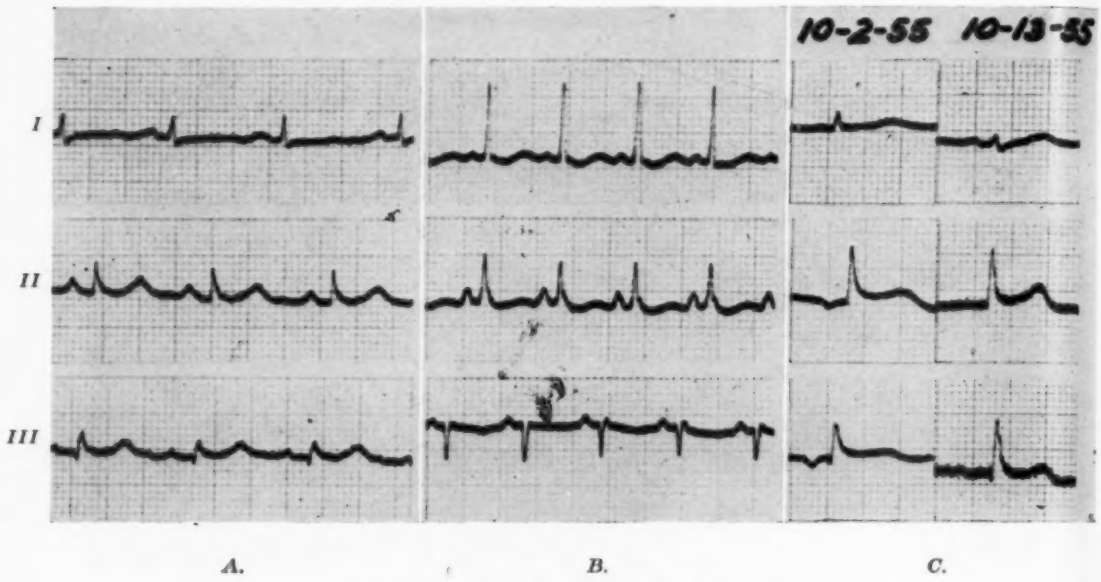


**Fig. 3.—Standard leads in three different patients with tetanus.**

## DISCUSSION

Tetanus is a disease in which a powerful toxin is liberated at the site of entry and carried through the blood stream and lymphatics to all the tissues of the body. It is the effect of this toxin upon the central nervous system and the neuromuscular junctions that brings about the clinical picture of the disease.[15,16]

The effect of this toxin upon the cardiac muscle has not been previously studied. The difficulty in obtaining adequate tracings in the presence of severe muscular contractions probably accounts for the lack of electrocardiographic exploration in tetanus.

The use of self-retaining electrodes as well as the elimination of the electrocardiographic distortion due to somatic tremors has made it possible for us to study the electrocardiographic changes in 14 cases of generalized tetanus.

Some of the findings encountered are insignificant. The juvenile pattern, found in some cases, is normal in young patients and some adults, and no clinical importance can be attributed to it.[17] Sinus arrhythmia is also a normal phenomenon of no clinical significance.[18]

Although sinus tachycardia may in part be explained on basis of fever in three instances, the remaining three patients were afebrile. In the latter, the tachycardia is attributed to the stress of the disease.

The remaining electrocardiographic alterations, although nonspecific, are suggestive of some effect of this disease upon the heart. Low QRS amplitude occurred in one instance. As there was no evidence of associated electrical insulating factors such as edema, emphysema, obesity, etc., we are led to postulate that it was most likely due to the action of the toxin upon the myocardium. This same mechanism probably accounts for the prolongation of the Q-T interval in five instances and the P-wave changes in two. In the latter, the effect of the toxin was apparently more marked upon the auricular muscle.

Nodal tachycardia, premature auricular beats, and premature ventricular beats were registered in some of the cases. Their frequency is not high enough to warrant any conclusions; however, it is open to speculation whether the toxin is able to increase the irritability of different foci of the heart.

The S-T elevation may be explained on the basis of further damage upon the heart brought about by the tetanus toxin leading to myocardial injury.

As the changes previously described are transitory, it has to be postulated that apparently the tetanus toxin is able to cause biochemical or neurogenic alterations in the cardiac muscle that lead to electrocardiographic abnormalities. A careful histopathologic study of the hearts from patients dying from the disease should be performed in order to determine whether anatomical alterations are also produced.

## SUMMARY

The electrocardiographic findings in 14 patients with generalized tetanus have been presented. In order to obtain these tracings, self-retaining electrodes applied to the shoulders and inguinal regions were used. Abnormal but nonspecific electrocardiographic alterations were obtained. Their possible explanation has been discussed.

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# FREQUENCY OF RHEUMATIC HEART DISEASE IN MIAMI, FLORIDA: AUTOPSY FINDINGS

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ACCURATE incidence and prevalence rates pertaining to rheumatic heart disease are difficult, if not impossible, to obtain. Criteria for diagnosis as well as interpretation of clinical findings are flexible, and comparisons of data from different sources emphasize these variations. In a study of the files of outpatients seen at the clinic of the National Children's Cardiac Hospital, many conditions were confused clinically with rheumatic disease,[1] further indicating actual problems that arose when true evaluation of rheumatic incidence was desired. In order to judge how rheumatic rates may vary in different environments, information must be obtained by as many methods as possible.

In 1950, Saslaw, Ross, and Dobrin[2] reported the results of an investigation in Dade County, Florida, in which they stated that 3 to 5 of 1,000 native-born children examined while attending the local public schools were found to have auscultatory evidence of valvular involvement. Later evidence[3] was presented to demonstrate the lower frequency of rheumatic disease in native-born Floridians as compared with non-Floridians. The possible climatic influence on this rate was stressed.

Companion investigations were undertaken to add further knowledge to incidence and prevalence data already reported. Hereditary and environmental factors are under study.[4] The present approach was designed to determine the frequency of rheumatic heart disease in post-mortem examinations in Dade County, Florida, and to compare this finding with similar reports from other laboratories.

## METHOD

All reports of autopsies performed at Jackson Memorial Hospital, Miami, Florida, from Jan. 1, 1950, through Dec. 31, 1955, were reviewed for evidence of rheumatic heart disease. The cases were listed as showing rheumatic involvement according to the following criteria, described by Wallach, Lukash, and Angrist[5]: (1) fusion and retraction of the mitral or tricuspid valve; (2) fusion and shortening of the chordae tendineae; (3) noncalcific fusion of the aortic valve; (4) fusion of the aortic valve, with or without calcification, when associated with definite rheumatic mitral valve involvement; (5) Aschoff bodies or verrucae characteristic of active rheumatic disease; (6) mitral or aortic bacterial endocarditis superimposed upon previously distorted valves.

Excluded from the rheumatic classification were those with: (1) slight mitral changes not

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fulfilling points (1) or (2) above; (2) slight aortic stenosis; (3) calcific aortic stenosis; (4) calcification limited to valve rings.

Those specimens which met the above standards were tabulated, together with other pertinent information: (1) total number of patients discharged from the Hospital; (2) total number of deaths; (3) total number and percentage of autopsies; (4) average age of death of rheumatic patients.

In addition, comparable statistics were accumulated for all deaths at the Hospital, and also for all deaths in Dade County, Florida.

## RESULTS

Table I shows that 2.5 per cent (75 of 2,967 autopsies) of the examinations revealed the presence of rheumatic carditis. Only 52 (1.7 per cent) died of rheumatic disease or its complications (congestive heart failure, bacterial endocarditis, or embolism).

Comparative data reported in other studies are listed in Table II, arranged according to the year of the report. Correlation of the autopsy rate with the latitude of the area of each report was attempted, and weighted averages were calculated also but failed to show any relationship.

TABLE I. MORBIDITY AND MORTALITY DATA
GENERAL AND RHEUMATIC HEART DISEASE (1950-1955)

| | 1950 | 1951 | 1952 | 1953 | 1954 | 1955 | TOTALS |
|---|---|---|---|---|---|---|---|
| Number of discharges | 20,966 | 20,654 | 21,654 | 25,134 | 27,117 | 29,776 | 145,301 |
| Number of deaths | 970 | 959 | 936 | 1,130 | 1,143 | 1,220 | 6,358 |
| Autopsies Number | 499 | 465 | 410 | 498 | 478 | 617 | 2,967 |
| Autopsies Per cent | 50.5 | 48.5 | 43.8 | 44.1 | 41.8 | 50.6 | 46.7 |
| Number of birthplaces recorded | 487 | 452 | 405 | 479 | 468 | 608 | 2,899 |
| Total Florida-born Number | 112 | 132 | 94 | 98 | 142 | 147 | 725 |
| Total Florida-born Per cent | 23.0 | 29.2 | 23.2 | 20.5 | 30.3 | 24.2 | 25.0 |
| R. H. D. Florida-born | 1 | 1 | 1 | 0 | 0 | 3 | 6 |
| Average age | 59 | 7 | 29 | | | 25.3 | 28.5 |
| R. H. D. White | 17 | 15 | 9 | 2 | 6 | 15 | 64 |
| R. H. D. Negro | 3 | 3 | 3 | 0 | 1 | 1 | 11 |
| R. H. D. total Number | 20 | 18 | 12 | 2 | 7 | 16 | 75 |
| R. H. D. total Per cent | 4.0 | 3.9 | 2.9 | 0.4 | 1.5 | 2.6 | 2.5 |
| Average age | 54.4 | 46.5 | 51.0 | 46.5 | 50.3 | 49.9 | 50.4 |
| Deaths due to R. H. D. | 14 | 14 | 9 | 1 | 5 | 9 | 52 |
| Average age | 50.1 | 43.4 | 45.0 | 35.0 | 44.6 | 47.7 | 46.2 |

The average age of the 75 rheumatic patients at time of death was 50.4 years (Table I), while the 52 who actually died of the disease or its complications averaged 46.2 years. These average ages were compared with the average age at death of the 6,181 patients who died at the hospital during the period of the study (Table III), and on whom this information was available. The average age at death was 51.1 years. However, since rheumatic fever is exceedingly rare under 1 year of age, the number of deaths of newborn infants and infants under 6 months of age was deducted, and the age at death was recalculated. The corrected average age of 5,375 patients ending in death was 58.7 years.

TABLE II. REPORTED FINDINGS OF RHEUMATIC HEART DISEASE IN POST-MORTEM STUDIES

| YEAR OF PUBLICATION | LOCATION | REPORTED BY | NUMBER OF CASES | PER CENT R. H. D. | LATITUDE |
|---|---|---|---|---|---|
| 1924 | Boston | Harrison and Levine[6] | 1,362 autopsies | 4.69 | 42° N |
| 1924 | Boston | Harrison and Levine[6] | 1,733 autopsies | 3.45 | 42° N |
| 1924 | Baltimore | Harrison and Levine[6] | 6,911 autopsies | 1.30 | 39° N |
| 1924 | New Orleans | Harrison and Levine[6] | 2,552 autopsies | 0.23 | 30° N |
| 1924 | Oklahoma City | Harrison and Levine[6] | 383 autopsies | 0.00 | 35° N |
| 1927 | Galveston | Stone and Vanzant[7] | 210 autopsies | 4.3 | 29° N |
| 1931 | Boston | Davis and Weiss[8] | 5,215 autopsies | 9.3 | 42° N |
| 1931 | Cincinnati | Glazer[9] | 2,344 autopsies | 2.6 | 39° N |
| 1931 | Baltimore | Longcope[10] | 8,164 autopsies | 1.6 | 39° N |
| 1936 | Miami | Nichol[11] | 401 autopsies | 4.0 | 25° N |
| 1942 | San Jose, Costa Rica | Garcia Carillo[12] | 1,000 autopsies | 1.1 | 10° N |
| 1943 | Buenos Aires | Cossio[13] | 10,000 cardiacs | 18.2 | 35° S |
| 1945 | San Juan, P. R. | Suarez[14] | 1,259 autopsies | 2.1 | 19° N |
| 1945 | New Orleans | Uyeda[15] | 12,317 autopsies | 1.35 | 30° N |
| 1946 | Johannesberg | Becker[16] | 3,000 autopsies | 3.5 | 26° S |
| 1946 | Curacao, N. W. I. | Hartz and van der Sar[17] | 1,307 autopsies | 1.5 | 12° N |
| 1947 | New Orleans | Holoubek and Holoubek[18] | 8,313 autopsies | 1.4 | 30° N |
| 1947 | Mexico City | Robles Gil[19] | 750 rheumatics | 11.0 | 20° N |
| 1948 | Cleveland | Wartman and Hellerstein[20] | 2,000 autopsies | 6.0 | 42° N |
| 1950 | Winston-Salem | McMillan and Jones[21] | 582 autopsies | 9.27 | 37° N |
| Present | Miami | Saslaw and Johnson | 2,967 autopsies | 2.5 | 25° N |

TABLE III. AGE AT DEATH, JACKSON MEMORIAL HOSPITAL (1950-1955)

| | 1950 | 1951 | 1952 | 1953 | 1954 | 1955 | TOTALS |
|---|---|---|---|---|---|---|---|
| Number of deaths, age recorded | 934 | 907 | 884 | 1,117 | 1,130 | 1,209 | 6,181 |
| Average age | 49.0 | 50.1 | 49.8 | 52.1 | 51.4 | 53.0 | 51.1 |
| Deaths, newborn and under 6 months | 135 | 122 | 121 | 131 | 151 | 146 | 806 |
| Deaths, over 6 months | 799 | 785 | 763 | 986 | 979 | 1,063 | 5,375 |
| Average age, corrected | 57.3 | 57.9 | 57.6 | 59.0 | 59.4 | 60.3 | 58.7 |
| R. H. D., average age | 50.1 | 43.4 | 45.0 | 35.0 | 44.6 | 47.8 | 46.2 |

TABLE IV. MORTALITY STATISTICS FOR DADE COUNTY, FLORIDA (1950-1955)

| AGE (YEARS) | 1950 | 1951 | 1952 | 1953 | 1954 | 1955 | NUMBER OF DEATHS |
|---|---|---|---|---|---|---|---|
| Under 1 | 320 | 320 | 359 | 365 | 436 | 449 | 2,249 |
| 1– 4 | 58 | 51 | 65 | 55 | 72 | 56 | 357 |
| 5– 9 | 16 | 26 | 30 | 33 | 34 | 26 | 165 |
| 10–14 | 9 | 17 | 20 | 20 | 16 | 12 | 94 |
| 15–19 | 28 | 31 | 40 | 22 | 23 | 36 | 180 |
| 20–24 | 39 | 41 | 57 | 37 | 60 | 40 | 274 |
| 25–34 | 172 | 154 | 182 | 190 | 188 | 210 | 1,096 |
| 35–44 | 349 | 375 | 349 | 386 | 345 | 414 | 2,218 |
| 45–54 | 693 | 705 | 751 | 849 | 786 | 829 | 4,613 |
| 55–64 | 992 | 1,050 | 1,157 | 1,150 | 1,242 | 1,347 | 6,938 |
| 65–74 | 1,032 | 1,150 | 1,293 | 1,392 | 1,418 | 1,634 | 7,919 |
| 75+ | 908 | 1,004 | 1,123 | 1,229 | 1,314 | 1,482 | 7,060 |
| Totals | 4,616 | 4,924 | 5,426 | 5,728 | 5,934 | 6,535 | 33,163 |

TABLE V. AGE AT DEATH, DADE COUNTY, FLORIDA (1950-1955)

| | 1950 | 1951 | 1952 | 1953 | 1954 | 1955 | TOTALS |
|---|---|---|---|---|---|---|---|
| Total deaths | 4,616 | 4,924 | 5,426 | 5,728 | 5,934 | 6,535 | 33,163 |
| Average age | 56.4 | 56.5 | 57.1 | 57.7 | 57.4 | 58.2 | 57.4 |
| Deaths under 1 ÷ 2 | 160 | 160 | 179.5 | 182 | 218 | 224.5 | 1,124.5 |
| Deaths over 1 + ½ under 1 | 4,450 | 4,764 | 5,246.5 | 5,546 | 5,716 | 6,310.5 | 32,038.5 |
| Average age | 58.5 | 58.4 | 59.0 | 59.6 | 59.6 | 60.3 | 59.4 |

TABLE VI. AGE AT DEATH, RHEUMATIC HEART DISEASE, JACKSON MEMORIAL HOSPITAL, MIAMI, FLORIDA (1950-1955)

| AGE (YEARS) | 1950 | 1951 | 1952 | 1953 | 1954 | 1955 | TOTALS |
|---|---|---|---|---|---|---|---|
| 0-10 | | 1 | | | | | 1 |
| 11-20 | | | | | | 1 | 1 |
| 21-30 | 2 | 2 | 1 | | | 2 | 7 |
| 31-40 | 3 | 3 | 4 | 1 | 2 | 1 | 14 |
| 41-50 | 3 | 4 | 1 | | 2 | 5 | 15 |
| 51-60 | 6 | 5 | 2 | 1 | 2 | 2 | 18 |
| 61-70 | 2 | 3 | 1 | | | 5 | 11 |
| 71-80 | 4 | | 2 | | 1 | | 7 |
| Over 80 | | | 1 | | | | 1 |
| Totals | 20 | 18 | 12 | 2 | 7 | 16 | 75 |

TABLE VII. CAUSE OF DEATH OF 75 RHEUMATIC PATIENTS IN DADE COUNTY, FLORIDA

| | 1950 | 1951 | 1952 | 1953 | 1954 | 1955 | TOTALS |
|---|---|---|---|---|---|---|---|
| *Rheumatic* | 15 | 15 | 12 | 2 | 7 | 11 | 62 |
| 1. Pneumonia | | | 1 | | 2 | 2 | 5 |
| 2. Pul. Atl. | | 1 | | | | | 1 |
| 3. Infarction | | | | | | | |
| a. Pulmonary | 1 | 1 | 2 | 1 | 0 | 3 | 8 |
| b. Cerebral | 4 | 0 | 0 | 1 | 2 | 2 | 9 |
| c. Multiple | 1 | | | | | | 1 |
| 4. S.B.E. | 3 | 2 | 1 | 0 | 1 | 2 | 9 |
| 5. C.H.F. | 5 | 8 | 5 | 0 | 2 | 2 | 22 |
| 6. Active R.H.D. | 1 | 3 | 3 | | | | 7 |
| *Nonrheumatic* | 5 | 3 | 0 | 0 | 0 | 5 | 13 |
| 1. Gastrointestinal | 3 | | | | | 3 | 6 |
| 2. Myo. I. | | 1 | | | | | 1 |
| 3. Renal | 1 | 1 | 0 | 0 | 0 | 1 | 3 |
| 4. Sicklemia | | | | | | 1 | 1 |
| 5. Lung abscess | 1 | | | | | | 1 |
| 6. Meningitis | | 1 | | | | | 1 |
| Totals | 20 | 18 | 12 | 2 | 7 | 16 | 75 |

Pul. Atl. = Pulmonary atelectasis.
S.B.E. = Subacute bacterial endocarditis.
C.H.F. = Congestive heart failure.
Myo. I. = Myocardial infarction.

Similar data were collected for all deaths in Dade County, Florida. Official vital statistics are maintained in categories of years as follows: under 1, 1-4, 5-9, 10-14, 15-19, 20-24, 25-34, 35-44, 45-54, 55-64, 65-74, 75 and over (Table IV). No average age of death was available, so that such a figure was calculated by using the middle year in each group (i.e., 1, 2.5, 7, 12, 17, 22, 30, 40, 50, 60, 70, and 80). The over-all average was 57.4 years, which, when corrected for the deaths of newborn infants and infants under 6 months, rose to 59.4 years (Table V). Table VI shows the distribution of deaths of rheumatic patients tabulated against age, while Table VII indicates the cause of death in each of the rheumatic patients.

## DISCUSSION

Autopsy data on occurrence of a disease is theoretically the most accurate method of estimation of frequency available. Unfortunately, many factors influence statistical analyses based on post-mortem examinations. Interpretation of autopsy data depends on adequate evaluation of each of the factors.

Some of the factors which influence autopsy rates are:

1. Criteria used for pathologic diagnosis. Obviously, the number of hearts listed as rheumatic will be greater if the pathologist considers minimal distortion such as valve-edge thickening as positive for the disease.

2. Economic strata from which the hospital population is drawn.

3. Year or years of study. Years of stress, economic, geographic, or other, may increase the ratio of rheumatic deaths.

4. Climate. Warmer climates are characterized by less clinical rheumatic fever than colder regions. Lower rheumatic rates at autopsy therefore may be anticipated in the lower latitudes. Altitude, rainfall, diurnal temperature variation, wind velocity, humidity, and the other elements of the climatic environment modify the incidence of the disease.

5. Nativity. A person may develop rheumatic disease in an area of high incidence, and then move to a more favorable location. Regardless of the benefit he may derive from his transposition, and regardless of the possible prolongation of life, whenever he dies, his autopsy will reveal the presence of rheumatic heart disease, and will become a statistic against the favorable location. When compared with the unfavorable region, the finding will actually show a much better figure for the very area of origin of the disease.

How may we interpret the findings in Dade County, Florida, after due consideration of the factors influencing autopsy rates of rheumatic heart disease? Deaths of Florida-born patients numbered only 6 (8 per cent of the total rheumatic deaths), at an average age of 28.5 years. In contrast, 25 per cent of the total number of autopsies were performed on people who had been born in this state. Thus, rheumatic disease was found only one-third as frequently in Floridians as in those born elsewhere. In this region, where rheumatic activity is recognized infrequently, the death rate of 2.5 per cent must be closely surveyed. The criteria employed by us were those described by Wallach, Lukash, and Angrist.[5] Since Jackson Memorial Hospital provides facilities for private, part-pay, and non-pay patients, the population served cuts across all economic levels, both white and Negro. The data collected, therefore, appear to consti-

tute a truly representative sample of the community, from the economic standpoint.

The present investigation encompasses a 6-year period, sufficiently long to obviate minor annual variations. The average age at death of the rheumatic individuals was 46 years. The average year of birth was about 1907. At the age of highest incidence of rheumatic fever, these people were children of World War I. The average age of death of the 6 rheumatic individuals born in Florida was 28.5 years; their average birth year was 1925; their critical period for rheumatic development was the height of the economic depression.

Place of birth and climatological area in which childhood was spent are of greatest significance in Florida, to which state many people have migrated, bringing their illnesses with them. Only 2 of the 6 Floridians were natives of Dade County. One was a 19-year-old white man and the other a 7-year-old Negro girl. Although absolute evaluation of the effects of climate is impossible in south Florida at the present time because few adults are native-born, the evidence favors strongly the tenet that native-born individuals show little rheumatic cardiac involvement. By far, most of the examinations which showed rheumatic stigmata were in those whose illness began in other regions.

## SUMMARY

Between 1950 and 1955 inclusive, 145,301 patients were admitted to Jackson Memorial Hospital, Miami, Florida. Of these, 6,358 died, and 2,967 (46.7 per cent) were autopsied. There were 75 (2.5 per cent) whose hearts showed rheumatic disease, as judged by the criteria of Wallach, Lukash, and Angrist. Only 52 (1.7 per cent), however, died of rheumatic heart disease or its complications.

Data are presented for all deaths at Jackson Memorial Hospital, as well as information relative to the total deaths for Dade County, Florida.

Of 2,899 autopsies in which birthplace was recorded, 25 per cent (725) listed their nativity as Florida. Only 8 per cent (6) of the autopsies showing rheumatic disease were among Florida-born individuals. Floridians in this series demonstrated a mortality rate from rheumatic fever and rheumatic heart disease of one-third the over-all death rate.

Death rates for rheumatic heart disease are compared with similar findings elsewhere in the world. Factors which influence the rate are discussed. These factors include the criteria used for pathologic diagnosis; the economic strata from which the hospital's population is drawn; the years of study as affected by unusual economic, martial, or other critically stressful situations; the climate of the area in which the individual spent his years of childhood, when most prone to rheumatic attack; and, finally, the corollary effects of latitude, altitude, temperature, humidity, barometric pressure, diurnal change in temperature, and so forth. How these factors apply in the Miami area is indicated.

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# A REVIEW OF 3,369 ELECTROCARDIOGRAMS IN YOUNG MALE HOSPITALIZED PATIENTS

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THERE have been relatively few studies on electrocardiographic variations in young people. Furthermore, a review of electrocardiograms in a young male hospitalized group has, as far as we know, not previously been made. A military hospital, where the patient population is, in major part, in the age group between 18 and 30, is an ideal place to carry out such an analysis. We have consequently reviewed all the electrocardiograms taken at the 3650th USAF Hospital, Sampson Air Force Base, during the years 1951 to 1955.

The 3650th USAF Hospital is an 1,100 bed general hospital. During the years 1951 to 1955, approximately 100,000 patients were admitted for medical care (all services, medical, surgical, neuropsychiatric, etc.). Tracings of males in the age group 17 to 24 years were considered. They were reviewed by the three authors independently, and when any disagreement concerning a tracing was present, its was reread in conference and a diagnosis was reached. No attempt was made to correlate the electrocardiographic diagnosis with the clinical diagnosis, because many charts, particularly in the years 1951 and 1952, were incomplete and almost fragmentary. During these years the hospital had just begun to function as a unit, and laboratory examinations were still at a minimum. A total of 8,000 patients with electrocardiograms were screened. Of those in the hospital population, 3,369 were in the age group in which we were interested. Of that total number, 289 (8.5 per cent) showed some abnormality. The abnormalities were divided into five categories (see Table I) and they will be discussed below.

TABLE I

| YEAR | TOTAL PATIENTS WITH TRACINGS (AGE 17-24) | CONDUCTION DEFECT | | ARRHYTHMIAS | DEXTROCARDIA | S-T SEGMENT + T-WAVE ABNORMALITY | ABNORMAL P WAVES + QRS COMPLEXES |
|---|---|---|---|---|---|---|---|
| | | P-R INTERVAL | RBBB | | | | |
| 1951-1952 | 1,168 | 37 | 7 | 24 | 2 | 38 | 0 |
| 1953 | 650 | 12 | 3 | 9 | 0 | 26 | 6 |
| 1954 | 804 | 24 | 7 | 4 | 0 | 11 | 2 |
| 1955 | 747 | 41 | 16 | 12 | 2 | 10 | 6 |
| Totals | 3,369 | 114 | 33 | 49 | 4 | 85 | 14 |

I. *Conduction Defects.*—

a. *P-R interval:* The top normal P-R interval was considered to be 0.20 second. When a question of correlation with a rate arose, the table of Ashman and

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Hull was consulted. Of the 289 abnormal electrocardiograms, 114, or 39 per cent, showed this prolongation. This represented 3.1 per cent of the total 3,369 electrocardiograms examined. Although it was not possible to correlate each chart with the abnormal electrocardiogram, our impression, clinically, was that many but not all these patients had a confirmed diagnosis of rheumatic fever. Infectious diseases such as measles, meningitis, hepatitis, and infectious mononucleosis were also represented.

b. *Bundle branch block:* It is interesting that no cases of left bundle branch block were present. Of the 289 abnormal tracings, 33 cases of right bundle branch block were discovered. This represented 11.4 per cent of the abnormal tracings, or less than 1 per cent of the total tracings in the group. The cases of right bundle branch block will be further commented upon in another communication.

II. *Arrhythmias.*—Forty-nine cases of arrhythmias were present in this series. This represented 16.9 per cent of the abnormal tracings, or 1.4 per cent of the total number in the series. There was 1 case of sinus tachycardia (rate 162), 1 case of sinus bradycardia, 1 case of many premature auricular contractions, 2 cases of paroxysmal auricular tachycardia, 1 case of auricular flutter, 1 case of auricular fibrillation, 3 cases of heart block (Wenckebach type), 8 cases of wandering pacemaker, 4 cases of frequent nodal extrasystoles, 7 cases of nodal rhythm, 2 cases of nodal tachycardia, 13 cases of frequent premature ventricular contractions, 1 case of ventricular tachycardia (patient recovered), 1 case of complete heart block (patient had rheumatic fever and recovered), 1 case of numerous blocked premature auricular contractions, and 2 cases of Wolff-Parkinson-White syndrome. Therefore, almost all types of arrhythmia were represented.

III. *Dextrocardia.*—In our series, 4 electrocardiograms were diagnosed as "dextrocardia." Of these, 2 patients had complete situs inversus, 1 patient had dextrocardia, and 1 patient had congenital absence of the right lung with mediastinal shift, so that this last case represents dextroposition, rather than true dextrocardia. In all of these patients no other cardiovascular abnormality was found.

IV. *S-T Segment and T-Wave Abnormality.*—There were 85 patients who showed some type of S-T segment and T-wave abnormality in Leads I, II, $aV_F$, or the precordial leads. This represented 29.7 per cent of the abnormal tracings, or 2.5 per cent of the entire series. Twelve of these patients were known clinically to have pericarditis.

V. *P Waves and QRS Complexes.*—In this category we included those patients whose electrocardiographic abnormality or abnormalities were confined solely to the P wave or QRS complex. Certainly there were other cases showing these abnormalities but they also had other pertinent findings and were therefore grouped in another category. The abnormalities were widening, notching, or peaking of the P wave and significant Q waves in the QRS complexes. There were a total of 14 such cases, 4.8 per cent of the abnormal tracings or 0.4 per cent of the total number of tracings.

## DISCUSSION

The age group of 17 to 24 years was chosen primarily because this is the group which is present in our induction centers and is the group with which the military is primarily concerned. Since this is apparently the first such survey on a hospital group, we thought it worth while to compare our figures with those of a similar age group, but individuals who were ambulatory and apparently in good health. We used for comparison two studies, one done by Ferguson and associates[1] in 1926, on 1,812 naval midshipmen, and the other done by Graybiel and associates[2] in 1942, on 1,000 naval pilots. Although the age groups were not exactly the same, they were similar and did overlap. Our figures show that in our hospitalized group 3.1 per cent of patients had abnormal P-R prolongation. In Ferguson's study the rate was 0.02 per cent. Our patients showed S-T segment and T-wave abnormalities in 2.5 per cent. In the Ferguson study 0.44 per cent showed abnormalities, and in Graybiel's study the incidence was negligible (0.003 per cent). In the other categories, our figures and those of Ferguson and Graybiel are not significantly different. No cases of dextrocardia were reported by them.

These figures deserve comment. Our patients were admitted with a variety of diagnoses. Many of these patients had an infectious disease of one type or another and many were febrile and toxic. The greater incidence of P-R prolongation and S-T segment and T-wave abnormalities in the hospital population as compared to a similar ambulatory population may simply be a reflection of the processes occurring in diseased states.

These results may further emphasize the fact that prolongation of the P-R interval, S-T segment variation (excluding known diagnosed pericarditis), and T-wave abnormalities are nonspecific and may represent the myocardial reaction to infection or inflammation. It should make the physician aware of the fact that these changes do not necessarily have to occur in patients who are suffering from primary cardiovascular disease, and should demonstrate the fact that the electrocardiogram is still a laboratory examination and does not make diagnoses. The great variations which occur must be realized and should always be appraised in terms of the entire clinical picture.

## SUMMARY AND CONCLUSIONS

1. A new study of 3,369 patients' electrocardiograms was made in a general hospital male population between the ages 17 to 24 years, of which 289 or 8.5 per cent were abnormal.

2. The two largest categories which showed abnormalities were prolongation of the P-R interval (3.1 per cent) and S-T segment and T-wave variation (2.5 per cent).

3. Our results in the above two particular categories show an increased incidence of abnormality as compared to two other studies done on apparently healthy ambulatory individuals in a similar age group. This observation may indicate that such electrocardiographic alteration represents nonspecific myocardial reaction to infection, inflammation, or toxic states.

4. The electrocardiogram in young hospitalized male patients may show considerable variation from the normal, and, consequently, it must be interpreted in conjunction with the entire clinical picture.

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# BLOOD PRESSURE AND THE WEATHER

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ALTHOUGH there has been speculation for centuries regarding the effects of weather on human health, there are few factual data in the medical literature regarding this intriguing subject. The excess of cardiovascular deaths in the winter months is one of the few well-established relationships, but whether the excess is due to increased metabolic demands, complicating respiratory infections, or more direct effects on the cardiovascular system is not known.

The pertinent studies on the relation of climate and weather to cardiovascular disorders fall into 3 general classifications. The first associated differences in cardiovascular mortality in various geographic areas to variations in climate.[1] However, Moriyama and Herrington[2] showed that when the cardiovascular mortality rates were adjusted for race and socioeconomic status, there was no increased mortality in areas with stormy, variable weather, as suggested by the earlier studies, although there was some increased mortality in areas with a cold, damp climate.

A second group of studies concerns the incidence of cardiovascular disorders as related to the passage of weather fronts. In Philadelphia, Newton[3] reported an increase in postoperative thromboses associated with cold fronts. Ströder, Becker, and Haas[4] in Germany, noted that on days with weather disturbances there was an excess of myocardial infarctions as compared to the number on days with stable weather conditions. Teng and Heyer[5] have also reported an increased incidence of myocardial infarctions in Texas, following the passage of cold fronts. The alterations in physiology which might be responsible for these effects associated with unstable weather are unknown, although Bean and Mills[6] suspect that the increased metabolic demands associated with lower temperatures may play an important part.

A third method of investigating this problem is to study the effects of weather or its components on the circulation in man. A number of studies of the effects of barometric pressure, temperature, and relative humidity have been reported by workers in the fields of industrial hygiene and aviation medicine, but these deal largely with extreme variations that are rarely encountered in ordinary experience. Among studies that report the effects of more usual variations is that of Scott, Bazett, and Mackie,[7] who investigated the changes in blood pressure associated with artificially produced changes in temperature and relative

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humidity. They concluded that warmth produced an initial rise and a later fall in blood pressure, and cold the opposite effect, but that the end result in both instances was an elevation in blood pressure over the readings at the beginning of the experimental period. It seems possible that the general tendency for an increase in blood pressure over the period of observation might have been related to experimental conditions other than the variables of temperature and relative humidity. This would appear to be an inherent difficulty in the experimental method.

Another approach would be to obtain measurements on a group of persons engaged in their usual activities on different occasions, and to ascertain whether circulatory changes appear to be related to changes in weather occurring over the interval between examinations. Such a study, using blood pressure determinations as the index of circulatory changes, was made possible by the activities of the Muscogee County Tuberculosis Study, a cooperative enterprise of the Muscogee County Health Department, Georgia State Health Department, and the U. S. Public Health Service.

## PROCEDURES

The present study was performed as a by-product of an investigation of blood pressure levels in a random sample of the residents of Muscogee County, Georgia, and of a concomitant investigation of the tuberculin sensitivity of an overlapping sample of adult residents of Muscogee County, Georgia, and Russell County, Alabama. Details of the projects were previously described.[8] Blood pressure determinations were made before and after the administration of a tuberculin test, and again 40 to 80 hours later when the tuberculin reactions were measured. The majority of the determinations were made in the subjects' homes. When this arrangement was inconvenient, the subjects were examined in the Health Center or at work, according to their wishes. Of the total group of 483 persons, 351 were white persons; and 321 were women. All but one were between the ages of 25 and 65 years.

The method of obtaining and recording blood pressure was that recommended by the American Heart Association.[9] The determinations were made by specially trained nurses, whose individual readings had been previously calibrated against those of each other to assure reasonable standardization of results. The readings made before the administration of the tuberculin tests, and those made at the time of the recording of the tuberculin tests (40 to 80 hours later) are used in the following comparisons. These are independent observations, since the examiner making the later blood pressure reading was not permitted to know the results of the previous examination. On each occasion, the blood pressure was taken three times in succession, but only the third observation was recorded.

Weather data were obtained from the U. S. Weather Bureau at the Muscogee County airport. Barometric pressure, air temperature, and relative humidity recorded at 2:30 P.M. were selected for study. This time was chosen because approximately half of the blood pressure determinations were made before, and half after, that time of day. Almost all were made within 4 hours of the recording of the weather observations. When weather fronts passed over the

area, the type of front and approximate time of passage were also furnished by the Weather Bureau. The great majority of the examinations were made in October and November, 1954. A few were made in July, September, and December, 1954, and a few in February and March, 1955.

## RESULTS

There was a general tendency for blood pressures observed on the days of the reading of the tuberculin test to be lower than those on the first examinations. It is believed that this decrease was the result of "desensitization" of the subjects to sphygmomanometry. There was no correlation of blood pressure change with degree of sensitivity to tuberculin.

The mean changes in systolic and diastolic blood pressure according to changes in the three weather variables—barometric pressure, air temperature, and relative humidity—are shown in Table I. It is readily apparent that there is no discernible correlation between changes in these weather factors and changes in either systolic or diastolic blood pressure.

TABLE I. MEAN CHANGES IN BLOOD PRESSURE WITH CHANGES IN WEATHER FOR 483 SUBJECTS EXAMINED TWICE AT INTERVALS OF 2 TO 3 DAYS

| CHANGE IN WEATHER FACTOR BETWEEN FIRST AND SECOND BLOOD PRESSURE EXAMINATION | | NUMBER OF PAIRED OBSERVATIONS | SYSTOLIC | DIASTOLIC |
|---|---|---|---|---|
| Total | | 483 | −5.5 | −1.8 |
| Barometric Pressure (in 1/100 inches of mercury) | +30 and over | 54 | −10.0 | −1.3 |
| | +29 to +10 | 95 | −4.3 | −3.0 |
| | + 9 to −9 | 231 | −5.7 | −1.7 |
| | −10 to −29 | 66 | −1.8 | −0.8 |
| | −30 and less | 37 | −6.2 | −2.2 |
| Temperature (in °F.) | +10 and over | 72 | −8.4 | −1.2 |
| | + 9 to 0 | 189 | −5.2 | −1.7 |
| | −0.1 to −9 | 116 | −4.7 | −3.7 |
| | −10 to −19 | 61 | −3.0 | 0 |
| | −20 and less | 45 | −7.7 | −0.7 |
| Relative Humidity (in percent) | +20 and over | 50 | −1.6 | −1.6 |
| | +19 to 0 | 147 | −7.6 | −2.6 |
| | −0.1 to −19 | 237 | −5.2 | −1.7 |
| | −20 and less | 49 | −4.9 | −0.1 |

The relationship of the passage of weather fronts between the first and last blood pressure determinations to the changes in blood pressure is shown in Table II. Unfortunately, most of the examinations were performed in periods of stable weather conditions. There was no significant change in blood pressure associated with the passage of a cold front. Although the passage of a stationary front was associated with a statistically significant increase in mean systolic pressure, the fact that there were only 19 such observations makes this relationship no more than suggestive.

TABLE II. MEAN CHANGES IN BLOOD PRESSURE FOR 483 SUBJECTS EXAMINED TWICE AT INTERVALS OF 2 TO 3 DAYS, ACCORDING TO WEATHER FRONT PASSAGE DURING INTERVAL

| TYPE OF FRONT PASSING BETWEEN FIRST AND SECOND BLOOD PRESSURE EXAMINATION | NUMBER OF PAIRED OBSERVATIONS | MEAN CHANGE IN BLOOD PRESSURE, IN MM. Hg. | |
|---|---|---|---|
| | | SYSTOLIC | DIASTOLIC |
| Total | 483 | −5.5 | −1.8 |
| Cold | 50 | −6.3 | −1.6 |
| Stationary | 19 | +4.0 | −1.7 |
| None | 414 | −5.8 | −1.8 |


## SUMMARY

The results of paired blood pressure observations taken 40 to 80 hours apart on 483 adult residents of a southern community are reported in relation to weather changes over this interval. No evidence could be found to indicate that changes in barometric pressure, air temperature, or relative humidity were associated with changes in blood pressure. The passage of a cold front was not associated with changes in blood pressure, but there was a suggestion that the passage of a stationary front might be related to an increase in systolic blood pressure.


The authors wish to thank the personnel of the U. S. Weather Bureau in Columbus, Georgia, and especially Mr. Thomas Floyd, for their courtesy and assistance with the collection of weather data for this study.

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# THE RESPIRATORY COMPLICATIONS OF CONGENITAL HEART DISEASE

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## INTRODUCTION

IN 1777, Sandifort[1] described what was probably the first case of the condition now called the tetralogy of Fallot. This patient was afflicted also with frequent respiratory infections. More recent writers[2-4] on cyanotic conditions with pulmonary stenosis do not include frequency of respiratory infections in the symptomatology. Mason,[5] however, in review of the surgical treatment of Fallot's tetralogy, states that precedent respiratory infections are common, but gives no details.

It is the purpose of this paper to describe the incidence of respiratory conditions in diverse types of congenital heart disease and to comment on some of their consequences.

## SOURCE OF DATA

The information presented is based upon a series of 173 consecutive cases of congenital heart disease from the pediatric service of the University of Wisconsin. The methods of diagnosis have already been presented.[6] The conclusions embodied in the present report have been confirmed by a later series from the same source.

## INCIDENCE

Respiratory infections are frequent in defects which are associated with a left-to-right shunt.[7] Table I confirms this, but also shows a high incidence in lesions associated with a true restriction of the pulmonary flow. It is emphasized that all types of respiratory infections except coryza are included. The impression remains that these infections are more severe in tetralogy, transposition of the great vessels, atrial septal defect, and ventricular septal defect in children less than 1 year of age. It is worthy of remark that children with "pure" pulmonary stenosis or the adult type of coarctation are relatively free of this complication. The high incidence in transposition reported here is not in accordance with the findings of Keith[8]; however, Taussig[2] states that pulmonary infections are common in this condition if the heart is greatly increased in size.

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TABLE I. INCIDENCE OF RESPIRATORY INFECTIONS IN CLINICAL HISTORY

| DISEASE | TOTAL | RESP. INF. | % |
|---|---|---|---|
| Tetralogy of Fallot | 33* | 18 | 54 |
| Transposition of great vessels | 13* | 8 | 61 |
| Tricuspid atresia | 6* | 2 | 33 |
| Pure pulmonary stenosis | 8 | 0 | 0 |
| Pulmonary stenosis, R-L shunt—normal aortic root | 5 | 2 | 40 |
| Coarctation, adult type | 10 | 0 | 0 |
| Coarctation with L-R shunt (A.S.D., V.S.D., P.D.A.) | 8 | 5 | 63 |
| Atrial septal defect | 41 | 27 | 69 |
| Ventricular septal defect | 22 | 13 | 59 |
| Patent ductus arteriosus | 27 | 18 | 66 |
| | 173 | | |

*Surviving three months or more.

Table II assesses the incidence of respiratory infections as the leading complaint. This is a measure of the importance of this complication to parents, who are commonly unaware of or not impressed by such "cardiac" complaints as cyanosis or moderate dyspnea. It is seen that the problem is as great in tetralogy as in atrial septal defect.

TABLE II. RESPIRATORY INFECTIONS AS A LEADING PARENTAL COMPLAINT

| LESION | TOTAL | PERCENTAGE |
|---|---|---|
| Tetralogy of Fallot | 33 | 20 |
| Atrial septal defect | 41 | 18 |
| Coarctation, adult type | 10 | 0 |
| Patent ductus arteriosus | 27 | 3 |
| Ventricular septal defect | 22 | 10 |
| Pure pulmonary stenosis | 8 | 0 |

Sources as in Table I.

## NATURAL HISTORY

Natural history is of considerable interest. It has already been shown how much importance parents attach to these infections—often to the exclusion of purely cardiac complaints.

Commonly, the patient begins with an ordinary "cold," with running nose and early onset of cough. Fever is often absent in the first 36 hours. The cough increases in severity and definite signs of bronchitis appear. Dyspnea begins or increases if already present. Cyanosis may be precipitated in a previously acyanotic tetralogy, or the well-known reversal of shunt may occur in those who suffer from pulmonary plethora. Antibiotic therapy and steam may abort the attack at this stage, but occasionally true consolidation will develop.

Laryngitis is rarely found; hoarseness or aphonia may be due to a recurrent laryngeal nerve palsy caused by compression between the pulmonary artery and the aorta. Six of the cases of atrial septal defect had this complication.

The frequency with which such infections follow each other is often remarkable. Thus, an infant with ventricular septal defect was described as having a continuous cough and respiratory infection! Similarly, a case of tetralogy had five severe respiratory infections in two months, three of these requiring hospitalization. In 1954, this patient spent 89 days in the hospital with respiratory infections alone.

The onset of the infections is sometimes early in infancy; the first attacks may have been suffered by the age of 6 weeks. However, a later onset at 3 to 4 months is more common. This holds for cyanotic and acyanotic cases. Early onset is particularly common in transposition, tetralogy, and ventricular septal defect with pulmonary hypertension. Usually patent ductus (except the "malignant" type) is much less troublesome in this respect.

The frequency of infection remains approximately the same during the first 2 to 3 years. Thereafter, a slow decline occurs though the incidence is probably greater still than in normal persons. The pattern is exemplified in the patient with a left-to-right shunt by a 15-year-old boy who had a ventricular septal defect. In his first 3 years, he had 10 to 12 attacks of bronchitis annually and at all seasons. By the age of 6, he suffered only in the fall and winter. At 10 years of age, he had only 1 or 2 attacks per year—usually of a minor nature.

In view of the paucity of untreated cases of tetralogy attaining riper years, numerous parallel examples cannot be quoted. However, a similar pattern has been observed in three 8-year-olds with tetralogy, and in another, aged 9, who had tricuspid atresia.

Two patients with transposition, aged 6 and 8 years, respectively, have had fewer infections in their later years. These are hardly typical however, being the sole survivors of a group of which the majority died in the first year.

It is of interest to speculate upon the etiology of these infections. Since it appears that patients with tetralogy and similar syndromes suffer almost as much as those with pulmonary plethora, it would appear that increased pulmonary blood flow is not the cause per se. Table II has shown the relative freedom in pulmonary stenosis without aortic displacement or septal defect. This lesion and aortic coarctation of the adult type represent the only common congenital defects in which the pulmonary blood flow remains intact and probably within normal limits. These had no more than the normal number of respiratory infections.

Table III gives some idea of the diversity of hemodynamics occurring within the group of patients with an unusual number of respiratory infections. There is a clear suggestion that the incidence is not related to the direction of the shunt, whether this is purely left-to-right, right-to-left, or bidirectional. Perusal of the pressure levels shows, as expected, that no relationship can be made out for this factor either. The only tenable deduction is that an increased or decreased pulmonary flow is associated with respiratory infections. This is not to say that either is the primary cause. We have noted severe respiratory infections in children with malignant patent ductus arteriosus or ventricular septal defect with pulmonary hypertension; however, the absence of basic criteria for the severity of such infections makes it difficult to delineate any relationship between these and raised pulmonary arterial pressure, whether as cause or as a result.

Exposure to sibling or school infection is a difficult factor to evaluate. It appears, however, that singletons are no less liable although their opportunities for cross-infection are less. Similarly, the observation of increased incidence of infections in the preschool child appears to reduce the relevance of school exposure. It should be remembered, however, that death and disability reduce the ranks of those who would attend school.

TABLE III. HEMODYNAMICS IN CONGENITAL HEART DISEASE WITH FREQUENT RESPIRATORY INFECTION

| LESION | AGE | CARDIAC OUTPUT (L./MIN.) | PULM. FLOW (L./MIN.) | L-R SHUNT (L./MIN.) | R-L SHUNT (L./MIN.) | MEAN P.A. PRESSURE (MM. Hg) |
|---|---|---|---|---|---|---|
| V.S.D. + coarctation | 6/12 | 1.4 | 3.3 | 1.87 | 0.1 | 67 |
| Tetralogy | 4 | 1.35 | 1.0 | 0.1 | 0.46 | 3.3 |
| A.S.D. | 4 | 3.9 | 7.3 | 3.4 | —— | 14 |
| P.D.A. | 4/12 | 1.6 | 2.1 | 0.5 | —— | 46 |
| A.S.D. + pulmonary stenosis (aorta normal) | 11/12 | 4.31 | 2.2 | —— | 2.1 | 10 |
| V.S.D. | 7/12 | 2.4 | 2.44 | .84 | .84 | 77 |
| V.S.D. | 1 | 1.63 | 2.2 | 0.57 | —— | 26 |
| P.D.A. | 9/12 | 1.2 | 0.9 | —— | 0.3 | 71 |
| P.D.A. | 2 | 1.8 | 3.5 | 1.7 | —— | 17 |
| P.D.A. | 11 | 2.7 | 1.6 | —— | 1.1 | 77 |

## COMPLICATIONS DUE TO RESPIRATORY INFECTIONS

Apart from the primary morbidity already noted, Table IV illustrates the clinical associations of respiratory infections in a small unselected group of tetralogies. As in adults with heart disease, e.g., mitral stenosis, lung infections are dangerous. It may be objected that the congestive failure observed in two of the patients was the cause of the respiratory infection and not its result.

TABLE IV. CLINICAL ASSOCIATIONS OF RESPIRATORY INFECTIONS IN TETRALOGY OF FALLOT

| AGE AT ONSET | EFFECT |
|---|---|
| 3 months | Induced congestive failure |
| 6 months | Led to discovery of congenital heart disease |
| 3 weeks | Precipitated cyanosis |
| 6 months | Precipitated cyanosis |
| 5 months | Intermittent cyanosis became constant |
| 3 weeks | Precipitated cardiac failure |
| 4 months | Syncopal attacks increased from 3 to 10 daily during infection |
| 3 months | Onset of permanent dyspnea |
| 6 months | Onset of paroxysmal dyspnea |
| 8 months | Followed by intracranial venous thrombosis |

However, we failed to observe the preliminary pathologic weight gain, which is the first definite sign of congestive failure, until the respiratory infection was clinically well established. Concomitant closure of a ductus as an associated cause of the failure was excluded by autopsy, which revealed a fibrosed liga-

mentum arteriosum, although this factor may have operated in other cases where autopsy was not done. Several of our patients with atrial septal defect and one patient with coarctation with ventricular septal defect died following an apparently mild respiratory infection. A similar exitus without apparent congestive failure has occurred in several cases of transposition of the major vessels.

## PROPHYLAXIS

Clearly, protection against pertussis should be carried out early in life if a congenital heart lesion is discovered. Similarly, on exposure to measles, attenuation or complete protection is rational.

By a process of "passive" transfer, the method of penicillin prophylaxis, as used in rheumatic fever, has been advised in these patients with congenital heart defects. Although this lacks the specificity of streptococcal prophylaxis, there is suggestive clinical evidence that severe respiratory infections may thus be reduced. The incidence of infections occurring in a normal community is so obscure that a control series is most difficult to construct and statistical proof is therefore lacking. The beneficial effect of antibiotics should therefore be assessed against the spontaneous decrease with age already mentioned. Thus, the evidence of decreased incidence is purely clinical, and not to be accepted as an established fact. In individual cases, it is, however, worthy of trial. The most satisfactory method so far used is an intramuscular injection of benzathine penicillin in a dose of 600,000 to 1,200,000 units once monthly.

## SPECIFIC TREATMENT

In the stage of fever and bronchospasm, management is similar to that in patients without heart disorder. In the cyanotic child, special care is necessary to maintain adequate hydration. This is especially so in small infants lacking sucking power. Here gavage is sometimes necessary to maintain adequate fluid and caloric intake. The frequency of antibiotic treatment raises the problem of insensitivity of the offending organism. This has not been so far a real problem, possibly because, as in infantile bronchiolitis, the specificity of antibiotics or chemotherapy has not yet been adequately demonstrated.

Oxygen therapy and mild sedation are useful adjuvants, and an increase in humidity may be helpful. Prophylactic digitalis in severely affected infants has not been used, but the infant should be observed for pathologic weight gain or other signs of congestive failure and appropriate therapy begun.

## SUMMARY

The morbidity of respiratory infections associated with congenital heart disease is presented and reviewed. It is found to be high in any lesion producing perversion of the pulmonary flow, whether as plethora or oligemia. The etiological background is briefly reviewed, and suggestions for prophylaxis and treatment are given.

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# CIRCULATORY ADAPTATIONS IN SPONGE FISHERMEN (SKIN DIVERS)

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ABOUT three hundred men living on the island of Kalymnos, Dodecanese (Greece), are professional sponge fishermen, working without special apparatus as skin divers off the coasts of Cyrenaiki and Tripolis in North Africa during the summer, and the coasts of the Dodecanese and Egypt during the winter time.

They work ten months a year, from 8 A.M. to 5 P.M. every day, i.e., during the time the sea bottom is illuminated by the sun, and one can discern the sponge colonies, found at a depth of 50 to 70 meters, as small gray spots.

The sponge fishermen dive continuously during the day's work, without taking any food in order to avoid vomiting and loss of efficiency. They perform 50 to 120 dives a day with an apnea lasting from one to two and one-half minutes.

Their ages range from 16 to 56 years, most of them being between 20 and 35 years old.

In an attempt to evaluate the circulatory adaptions in these persons, we have undertaken the present investigation.

Our attention was focused mainly on the right side of the heart which, in our opinion, must be influenced by such a long voluntary apnea during a great part of their lives.

## MATERIAL AND METHOD

Two groups were investigated at the island of Kalymnos, the first concerning 29 persons (October, 1955) and the second concerning 31 more (May, 1956).

Our study was limited for the first group—lacking x-ray apparatus on the island—to a clinical examination, and taking the blood pressure, twelve-lead electrocardiogram, and certain other measurements, i.e., age, years of work, and age at the beginning of the work. However, in the second group we have taken, besides the above, the heights, the weights, and a number of x-ray films in 9 persons of that group and in 5 persons of the first group who were re-examined.

## RESULTS AND DISCUSSION

Pertinent data of both groups are given in Tables I and II.

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TABLE I. FIRST GROUP OF SPONGE FISHERMEN EXAMINED AT KALYMNOS (OCTOBER, 1955)

| PATIENT NO. | AGE | YEARS OF WORK | B.P. (MM. Hg) | BEGAN WORK AT AGE | WEIGHT (KG.) | HEIGHT (CM.) | X-RAY TAKEN | PULSE RATE | AXIS | LEADS | | | | REMARKS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | I | III | $V_1$ | $V_2$ | |
| 1 | 18 | 1 | 130/80 | 17 | | | | 75 | N | | | R + 3 | R + 8 | |
| 2 | 45 | 13 | 130/80 | 32 | | | | 80 | −5° | | | R + 1 | R + 3 | |
| 3 | 30 | 3 | 135/90 | 27 | | | | 95 | O | | | R + 4 | R + 7 | |
| 4 | 31 | 10 | 135/80 | 21 | | | | 85 | N | | | R + ½<br>S 12 | R + 3<br>S 25 | |
| 5 | 20 | 2 | 120/80 | 18 | | | | 65 | N | R + 4<br>S 0 | R + 6<br>S 0 | R + 2 | R + 5 | |
| 6 | 48 | 30 | 130/80 | 18 | | | | 70 | N | | | R + 1<br>S 4½ | R + 6<br>S 8 | |
| 7 | 42 | 24 | 140/80 | 18 | 65 | 165 | + | 70 | N | R + 5<br>S 0 | R 2½ | R + 2 | R + 8 | |
| 8 | 55 | 38 | 135/80 | 17 | | | | 65 | −20° | | | R 0 | R + 2 | |
| 9 | 56 | 37 | 160/80 | 19 | | | | 65 | Mod. RAD | | | R 0 | R + 7 | |
| 10 | 20 | 3 | 130/80 | 17 | 65 | 168 | + | 65 | Sl. LAD | | | R + 2 | R + 6 | |
| 11 | 41 | 20 | 130/85 | 21 | 55 | 168 | + | 85 | N | R + 3<br>S 6 | R + 12 | R + 2 | R + 5 | |
| 12 | 19 | 2 | 130/75 | 17 | 53 | 166 | | 60–90 | 90° | | | R + 4 | R + 11 | Sinus Arrhythmia |
| 13 | 23 | 2 | 110/65 | 21 | 57 | 171 | | 70 | N | | | R + 2 | R + 10 | |
| 14 | 42 | 20 | 130/80 | 22 | | | | 80 | N | | | R + 2 | R + 7 | |

| | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 15 | 35 | 13 | 130/80 | 22 | | | | 70 | N | | | | | |
| 16 | 18 | 3 | 130/70 | 15 | | | | 90 | N | R + 4<br>S 1 | R = 0<br>S = 2 | R + 2 | R + 6 | |
| 17 | 26 | 6 | 140/80 | 20 | | | | 60 | N | | | R + 2<br>S 6 | R + 5<br>S 8 | |
| 18 | 21 | 5 | 130/80 | 16 | | | | 90 | N | R + 9<br>S 1 | Q − 1<br>R + 4 | R + 7 | R + 18 | |
| 19 | 27 | 6 | 110/70 | 21 | 67 | 173 | | 65 | Sl. LAD | Q + | | R + 4 | R + 6 | |
| 20 | 25 | 4 | 120/60 | 21 | 66 | 173 | + | 65 | N | | | R + 3<br>S 1 | R + 10 | Atrial septal defect |
| 21 | 19 | 5 | 125/75 | 14 | 57 | 168 | | 70 | N | R + 5<br>S 2 | R + 2<br>S 1 | R + 1½<br>S 3½ | R + 4<br>S 10 | Sl. wide $S_1$, wide R′ and VR |
| 22 | 47 | 16 | 120/80 | 31 | | | | 70–80 | Sl. LAD | R + 3<br>S 1 | R + 2½<br>S 0 | R + 2 | R + 6 | |
| 23 | 35 | 17 | 135/95 | 18 | 82 | 175 | | 80 | N | | | R + 1<br>prom. S | R + 4<br>prom. S | |
| 24 | 20 | 5 | 120/85 | 15 | 69 | 175 | | 90 | | R + 8 | | R + 4 | R + 7 | |
| 25 | 44 | 26 | 120/85 | 18 | | | | 75 | | L.V. | L.V. | R + 2 | R + 4 | Low voltage |
| 26 | 21 | 4 | 125/80 | 17 | | | | 80 | | | | R + 5 | R + 10 | |
| 27 | 39 | 23 | 130/70 | 16 | | | | 70 | | R + 5<br>S 0 | R + 7<br>S 0 | R + 2<br>S 7 | R + 3<br>S 8 | |
| 28 | 39 | 22 | 120/80 | 17 | 61 | 167 | + | 55 | | R + 5<br>S 1 | R + 2<br>S 0 | R + 3<br>S 4 | R + 5<br>S 15 | |
| 29 | 19 | 2 | 105/75 | 17 | | | | 55 | | Q 1<br>R + 8 | R ½<br>S 2 | R + 6<br>S 10 | R + 17<br>S 17 | |

TABLE II. SECOND GROUP OF SPONGE FISHERMEN EXAMINED AT KALYMNOS (MAY, 1956)

| PATIENT NO. | AGE | YEARS OF WORK | B.P. (MM. Hg) | BEGAN WORK AT AGE | WEIGHT (KG.) | HEIGHT (CM.) | X-RAY TAKEN | PULSE RATE | AXIS | REMARKS |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 31 | 10 | 120/80 | 21 | 60 | 170 | + | 90 | N | High $R_{V_2,V_3}$ |
| 2 | 23 | 2 | 110/70 | 21 | 70 | 176 | | 70 | N | High $R_{V_2\,V_3}$ |
| 3 | 26 | 10 | 120/75 | 16 | 60 | 164 | + | 60 | N | |
| 4 | 28 | 8 | 125/80 | 20 | 60 | 170 | | 65 | N | High $R_{V_2,V_3}$ but within normal limits (?) |
| 5 | 25 | 3 | 105/65 | 22 | 56 | 165 | | 55 | N | High $R_{V_2,V_3}$ but within normal limits (?) |
| 6 | 20 | 2 | 120/80 | 18 | 62 | 170 | | 65 | N | |
| 7 | 29 | 8 | 140/85 | 21 | 63 | 166 | | 55 | N | High $R_{V_2,V_3}$ but within normal limits (?) |
| 8 | 31 | 10 | 130/85 | 21 | 57 | 160 | | 40 | N | |
| 9 | 36 | 14 | 120/75 | 18 | 66 | 166 | + | 60 | N | |
| 10 | 35 | 14 | 140/80 | 18 | 88 | 177 | + | 70 | N | High $R_{V_1,V_2,V_3}$ |
| 11 | 25 | 10 | 125/75 | 15 | 68 | 169 | | 70 | N | |
| 12 | 26 | 7 | 130/80 | 19 | 58 | 165 | + | 80 | | Wide $S_{I,II,aVF}$ |
| 13 | 27 | 9 | 125/70 | 18 | 53 | 163 | | 75 | N | |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 14 | 25 | 1 | 135/80 | 24 | 64 | 171 | | 65 | N | |
| 15 | 19 | 3 | 100/70 | 16 | 59 | 168 | | 55 | N | Ventr. prem. beats |
| 16 | 21 | 1 | 140/80 | 20 | 63 | 167 | | 100 | N | |
| 17 | 27 | 8 | 130/75 | 19 | 63 | 170 | | 60 | N | |
| 18 | 31 | 15 | 130/80 | 16 | 73 | 179 | + | 100 | | Sl. wide $S_{I,II}$ |
| 19 | 28 | 9 | 130/80 | 19 | 57 | 165 | | 90 | N | |
| 20 | 41 | 20 | 110/70 | 21 | 62 | 173 | + | 75 | N | |
| 21 | 28 | 4 | 130/70 | 24 | 64 | 171 | | 85 | N | |
| 22 | 25 | 2 | 115/75 | 23 | 69 | 174 | | 85 | | High $R_{V_2,V_3}$ Atr. prem. beats |
| 23 | 42 | 20 | 120/70 | 22 | 75 | 178 | + | 75 | N | |
| 24 | 25 | 3 | 105/55 | 22 | 63 | 168 | | 90 | N | |
| 25 | 18 | 3 | 95/55 | 15 | 60 | 170 | | 65 | 90° | High $R_{V_1,V_2,V_3}$ |
| 26 | 26 | 9 | 110/60 | 17 | 65 | 172 | | 80 | N | |
| 27 | 17 | 1 | 120/70 | 16 | 55 | 160 | | 70 | | High $R_{V_2,V_3}$ |
| 28 | 21 | 2 | 125/80 | 19 | 56 | 166 | | 80 | N | |
| 29 | 25 | 1 | 135/70 | 24 | 58 | 162 | | 50 | N | |
| 30 | 44 | 27 | 110/80 | 17 | 62 | 163 | + | 65 | N | |
| 31 | 18 | 1 | 120/75 | 17 | 56 | 165 | | 70 | | WPW syndrome |

Clinical examination was negative in all cases except in Patient 20 of the first group, in whom a tentative diagnosis of an atrial septal defect was made on clinical, radiological, and electrocardiographic grounds, i.e., dyspnea on exertion, hyperdynamic right ventricle on palpation, systolic murmur at the pulmonary area, wide splitting of the pulmonary second sound, incomplete right bundle branch block in the electrocardiogram, and signs of pulmonary plethora on screening and in the x-ray film.

As a rule, the pulse rate and the blood pressure were found within normal limits. The electrocardiograms were found essentially normal with the usual run of mostly normal angles of the electrical axis and apparently normal ratio of right and left ventricles, and with the usual scattering of moderately pronounced left and right axis deviation dependent on the build of the subjects and the position of the heart resulting therefrom.

An "embryonic r" in Lead $V_1$ was present in a few patients (Nos. 12, 22, 26, 27, 29 of the second group), but this was thought to represent a normal finding.

In spite of disappointing weather, two experiments were performed on the same subject (Patient 4 of the second group), during which Lead $V_1$ was taken before and immediately after a dive of 55 and 60 seconds' duration. The T wave became inverted, the QRS complex showing no alteration. A 2:1 A-V block, observed after the second dive, disappeared after a minute.

## SUMMARY AND CONCLUSION

Clinical, electrocardiographic, and limited x-ray examination of two groups (29 and 31 cases) of sponge fishermen disclosed no abnormalities which could be attributed to an assumed strain on the right heart in these persons.

An entity of a physiologic cor pulmonale ("porpoise heart") may exist, although it was not apparent in this limited number of cases studied. It may be evident in rare cases of still more strain on the right heart from more prolonged underwater exertion. The problem deserves further study.

# STUDIES IN ANGIOCARDIOGRAPHY

## The Problems Involved in the Rapid, Selective, and Safe Injections of Radiopaque Materials. Development of a Special Catheter for Selective Angiocardiography.

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ENTHUSIASM about selective angiocardiography is rapidly increasing among heart physiologists and radiologists in this country.[1-3] Although the method was begun as far back as 1931, only in recent years is it being established as a reliable procedure. The techniques used in the early years were originated mainly as a means of obtaining x-ray pictures of the pulmonary vascular system.[4-7]

In 1947, in Mexico, Chávez, Dorbecker, and Celis,[8,9] performed successful selective angiocardiography. They used "direct intracardiac injections" of a radiopaque substance by means of a thick rubber catheter introduced via the jugular vein. The injections were made manually with an ordinary glass syringe, and because of the low-flow resistance of the catheter used, a great amount of radiopaque material could be rapidly injected. Thus an excellent contrast was obtained. At this time it was learned that when the injection was forceful, the catheter would recoil, displacing its tip, and thus interferring with selectivity in some cases, especially so when injections were attempted into the right ventricle.†[10]

In 1949, in Sweden, Jönsson, Brodén, and Karnell[11] succeeded in injecting a radiopaque material with the necessary rapidity by using Cournand-type catheters and a manually operated high-pressure injecting machine. The radiological contrast obtained in this series of selective angiocardiograms was outstanding. Selectivity was also improved, but not completely achieved in all cases, since they, too, found that when a forceful injection was made the catheter would recoil. They pointed out the dangers of producing disturbances of the heart rhythm due to the mechanical stimulation of the myocardium produced by the jet of radiopaque material coming from the tip of the catheter. This was particularly true when injections were made into the right ventricle.

In 1953, the Presoyector‡, a machine for automatic and safe high-pressure injections of radiopaque substance was developed and constructed by Rodríguez-Alvarez and Dorbecker at the Instituto Nacional de Cardiologia in Mexico City, Mexico. In working with this machine it was found that a more powerful injection was possible, but the same recoiling of the catheter was found to be a distressing problem.

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†The Nélaton catheters used had a lateral opening which gave a jet of liquid making an angle of approximately 30° with the catheter.

‡Registered trademark.

Experience with this machine and different catheters was presented by the same authors and Volnie in 1954.[12] In 1955, Rodríguez-Alvarez published some of the results obtained with an experimental catheter in which the hole at the tip had been occluded. Instead, several lateral openings were made in a zone 1.3 cm. above the tip. The openings were in pairs on opposing surfaces of the catheter, and the total surface of these openings was made as large as possible in order to diminish the lineal velocity of the jets. At the same time the recoiling of the catheter was markedly reduced.[13]

In 1956, in Sweden, Gidlund[14] presented a high-pressure injecting machine quite similar to the one described by the Mexican group in 1954.[15] With this machine he studied different aspects of injection. Using an artificial glass model of the heart chambers, he, too, concluded that in order to stop the recoiling of the catheter the use of lateral perforations was the answer.*

The problem of the rapid, selective, and safe injection of radiopaque materials has not been completely solved. Machines especially designed for high-pressure injection through cardiac catheters have become more simple, powerful, and safe in recent years. However, the ordinary catheters do not present the physical characteristics necessary to fulfill their duty of carrying a great amount of radiopaque liquid in a short period of time and with the desired degree of selectivity and safety. It is the purpose of this work to study some of the characteristics of different catheters and to present a special catheter developed by us in recent years, one which has been subjected to in vitro studies in order to evaluate quantitatively the recoiling impulse created during injection at different flow rates. Some important characteristics of this and other catheters will be presented.

In the early stages of our work in Mexico, and when the Presoyector was available, several attempts were made by one of us and Volnie[15] to use thin polyethylene catheters introduced percutaneously through a hypodermic needle. These experiments in dogs showed that the lack of radiopacity of these catheters could be easily overcome by slow, continuous injections of 30 per cent Diodrast. However, the catheter was difficult to manipulate making it almost impossible to place the tip of the catheter in the desired "organ host."† During injection the tip of the catheter would recoil greatly. These experiments demonstrated that the only advantage of these polyethylene tubes was their greater inner diameter and inner smooth surface‡ which permitted a great increase in flow. However, because of their other disadvantages the procedure was abandoned and Cournand catheters were used both in dogs and in men.

At this laboratory we have continued these experiments; our attention was devoted to improving this catheter to be used in selective angiocardiography and aortography. This work was started with the idea that such a catheter must fulfill at least the following characteristics: (1) It must be radiopaque. (2) It must have a wider inside diameter. (3) The inside and outside surfaces of the wall should be as smooth as possible. (4) It must have enough consistency in its wall to be easily manipulated. (5) Its tip must not recoil during injection even when a maximum flow is reached. (6) Although the radiopaque liquid may attain a high lineal velocity when traveling along the inside of the catheter, some means must be provided by the catheter to permit a slowing down of that velocity at the point where the liquid leaves the catheter at its distal end. (7) Even if a lesser degree of radiopacity of the catheter can be tolerated, the very tip of it, at least, must be highly radiopaque in order to be able to control the position of the tip with respect to the "organ host." (8) The wall must be able to withstand the high pressure used, especially at its proximal end. (9) The junction between the metallic connector and the catheter must be strong enough to withstand high pressure. (10) The wall must have the maximum strength that will assure against breakage or detachment, particularly at the portion of the tip where the lateral openings are to be located. (11) The catheter must be permanently curved near its tip. (12) It must be easily sterilized.

A catheter with all these characteristics has been devised and successfully manufactured.§

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*Other publications concerning these types of catheters have appeared recently.[16,17]

†"Organ host" is any segment of the circulation to be studied.

‡This became apparent after a comparative microscopic study made of a Cournand catheter and a polyethylene catheter.

§Made by U. S. Catheter and Instrument Corporation, Glenfalls, New York.

Some features deserve special mention because of the physical problems involved and because of their clinical importance. A smooth bore nylon core provided in this catheter has facilitated obtaining greater flow rates (Fig. 1). This nylon core also gives the catheter an extreme strength, making it able to withstand much more pressure than the regular catheters made in the past. These materials have given to its walls the desired consistency which makes it easy to manipulate.

The junction between the metallic tip and the catheter has been greatly improved. The radiopacity of the catheters has been slightly decreased, but a highly radiopaque tip has been achieved.

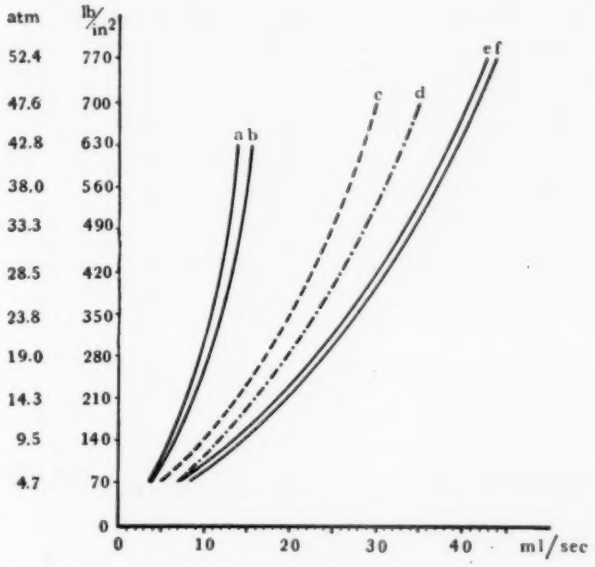


**Fig. 1.—Graph correlating actual pressure of injection in pounds per square inch or in atmospheres (roughly one atm. equals one kilogram per square centimeter) and flows in milliliters per second obtained through different catheters and with different fluids. (*a*) Cournand cardiac catheter 8F, 100 cm. long; fluid injected, water. (*b*) Lehman aortographic catheter 8F, 100 cm. long; fluid injected, water. (*c*) Catheter for selective angiocardiography and aortography devised by us, with 6 lateral openings, 8F, 100 cm. long; fluid injected sodium Urokon 70 per cent at a temperature of 21° C. (*d*) Same catheter as (*c*) with sodium Urokon 70 per cent but at 37° C. (*e*) Sample of a catheter constructed under the similar specifications, but with one terminal hole at the tip; fluid injected, water. (*f*) Same catheter as (*c*) and (*d*); fluid injected, water. All these injections were made into a flask, that is to say, against atmospheric pressure. We found before that there was not any important variation in the flow when injecting against a mercury column of 200 mm. using as an injecting machine a Presoyector.**

In a No. 8F catheter the outside diameter has been retained as in a standard No. 8F Cournand-type catheter, but it has been possible to increase the inside diameter from 1.32 mm. to 1.52 mm., thus the cross-sectional area of the lumen of this catheter has increased from 1.367 sq. mm. to 1.818 sq. mm., an increase of 33 per cent. The importance of this achievement will be realized later when we present the increases in flow obtained with this catheter as compared with the standard Cournand catheter of the same length and number.*

In a former publication the importance of diminishing the lineal velocity of the radiopaque substance at the tip of the catheter was analyzed briefly.[13] The average lineal velocity can be calculated in millimeters, if we know the flow in cubic millimeters per second and the cross-sectional area of the catheter in square millimeters:

$$V = F/A$$

Where V is average lineal velocity in millimeters per second, F is flow in cubic millimeters per sec-

*Catheters 7F, 6F, and 5F are now being manufactured.

ond, and A is cross-sectional area of the catheter in square millimeters in the case of a terminal hole at the tip or the sum of the areas of all the openings (as in the case of our catheter).

It is obvious that in order to diminish this lineal velocity we have to increase the area of ejection (i.e., the sum of all the areas of the openings from where the liquid leaves the catheter). Our catheter does not have a hole at the tip. In its place 6 lateral openings have been located in a zone 1.3 cm. immediately above the tip (Fig. 2). Each one of these openings has an area of 3.120 sq. mm. The total area of the 6 openings is 18.721 sq. mm. (that is to say, the total area of ejection) and it is 10.20 times greater than the cross sectional area of the catheter. If we apply the formula mentioned above, the average lineal velocity of each jet would be 10.2 times smaller than the average lineal velocity of a jet obtained with a catheter that would have the same cross-sectional area of our catheter, but with a single hole at the tip; average lineal velocities of 3 different catheters are shown in Fig. 3. This increase in area was not carried any further because of some physical problems encountered. We used a relatively small number of openings (6), but each one of them had a large area. We found this to be the best arrangement and the one which would not impair greatly the strength of the walls in this vital zone. (We already mentioned the possibility of breakage and detachment.) We did not choose the helicoidal arrangement of a great number of small openings because we found that this would weaken the strength of the walls along the helicoidal line formed by these openings. When using openings of a small area, many of them are needed in order to obtain a great total area of ejection; we achieved an area 10.2 times greater than the cross-sectional area of the catheter with only 6 openings.

**Fig. 2.—Tip of an 8F catheter for selective angiocardiography and aortography with 6 lateral openings. Actual size.**

Another problem was to locate the necessary number of openings which would give the required surface within a small zone of 1.3 cm. immediately above the tip. It was easier to fulfill this requirement by using very few openings of large surface than by using a great number of openings of small surface. This requirement of having all the openings within a small zone close to the tip is easily understood if we realize that in order to achieve selectivity we have to inject the radiopaque material into one chamber of the heart, or into one segment of the great vessels, at a time, otherwise more than one segment of the circulation will be opacified at the same time, thus losing selectivity.

In an ordinary catheter with a hole at the tip, an impulse is created during injection. This impulse can be expressed as a vector having the same direction as the velocity vector of the jet, but in the opposite sense.[19] Therefore, in our catheter we located our 6 openings, in opposing pairs, so that the impulse created at each opening would be counteracted by the one created at the opposite opening.

It could be predicted that with such a catheter the recoiling impulse would be at a minimum. In the case of an ordinary catheter with a hole at the tip, it would be easy to calculate the magnitude of this impulse, but in the case of our catheter with 6 lateral openings, there was no means of accurately calculating the resultant of these impulses.

It was realized that to measure these impulses was fundamental before attempting to evaluate our catheter and other catheters. An instrument for in vitro measurements of this force in dynes was devised and constructed by us at this laboratory using the principle that, when different tensions are applied to an elastic spring kept fixed at one of its ends, the displacement of the other

end where the forces are applied is proportional to these forces within the limits of elasticity of the spring. Fig. 4 shows the Presoyector,* the special dynamometer, and the catheter mounted on it, as used in these experiments. The outstanding details of this instrument are shown in Fig. 5. Tensions were produced in the spring (Fig. 5,*E*) by the application of from 1,500 to 90,000 dynes. Tensions could be changed at will during the experiment. A tension able to keep the catheter in place during injection was taken as the equivalent of force of the impulse. These tensions could be read on a fixed scale located under the spring (Fig. 5,*C*). Small weights 1 to 3 grams (Fig. 5,*L*) were added when necessary to counteract the elastic forces of the catheter tending to move it forward. It was absolutely impossible to avoid these forces, since a connection between the catheter and the Presoyector† had to exist at all times (Fig. 5,*K*). This precaution proved useful, as will be shown later.

The instrument was accurate from 1,500 to 90,000 dynes. The inertia of the movable parts of the system was kept at a minimum by making all parts from low-mass material (wooden rods, straight pins, and glass beads). The total mass of these parts amounted to 5.56 grams. We had no special interest in measuring forces below 1,500 dynes, since they were of the same order of magnitude as the elastic forces of the catheter itself and, therefore, difficult to control.[20]

In Fig. 6 a catheter No. 8F, devised by us, with lateral openings is shown when injecting at a flow rate of 27 ml. per second. Notice that the resultant of the recoiling impulses is not enough to counteract 3,000 dynes of tension given to the spring before the experiment (Fig. 6, *A* and *B*).

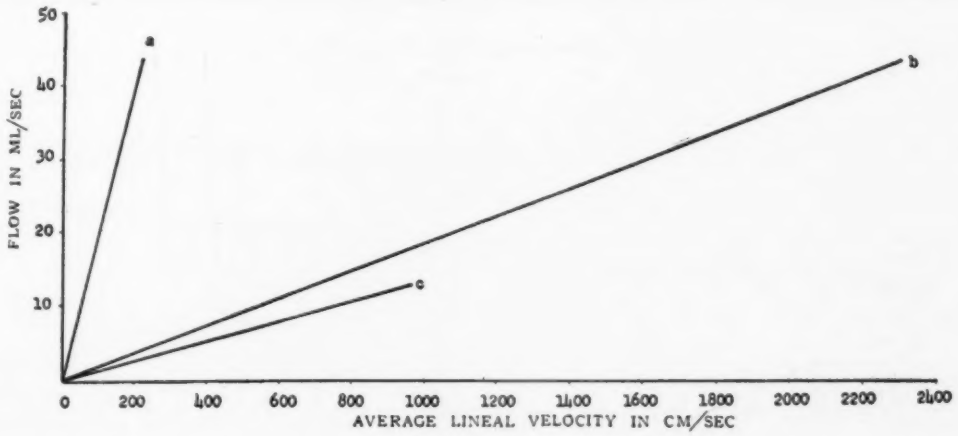


**Fig. 3.—Graph correlating flow in milliliters per second and average lineal velocity of the jet (or jets) in cm./second. (*a*) Catheter for selective angiocardiography and aortography with 6 lateral openings; size 8F, 100 cm. long. (*b*) Sample of catheter constructed under the same specifications as (*a*), but with one terminal hole at the tip. (*c*) Cournand cardiac catheter; size 8F, 100 cm. long. The important reduction in average lineal velocity of the jets for any given flow can be easily appreciated in the case of the catheter with lateral openings (*a*). The enormous average lineal velocity of a similar catheter with one terminal hole (having an area of ejection equal to the cross-sectional area of the catheter) may be seen in (*b*). (The average lineal velocity has been obtained by calculations.)**

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***In all these studies we used as an injecting mechanism a Presoyector to which some modifications were made; mainly, a syringe with a smaller diameter was used, the ratio between the area of the piston and the plunger of the syringe was thus raised from 4 to 1 in the prototype machine to 7 to 1 in this machine. Thus, using compressed air at a pressure of 110 pounds per square inch, pressure on the inside of the syringe may reach 770 pounds per square inch.**

**Flows obtained with each catheter at different pressures should be studied for each machine, since some physical characteristics may vary from one machine to another, even if made under the same specifications. Flow tables or graphs should be prepared for each catheter (Fig. 1), for each radiopaque substance, and for each temperature. For the benefit of a better understanding between different workers, the actual pressure of injection (the one obtained inside the syringe) should be used in these tables. Actual pressure of injection was calculated in our machine by multiplying the air pressure used by seven, which is the ratio between the areas of the piston of the cylinder and the plunger of the syringe.**

**†Obtainable from Presojector Mfg. Co. 1216 Pennsylvania Ave. Ft. Worth 4, Texas.**

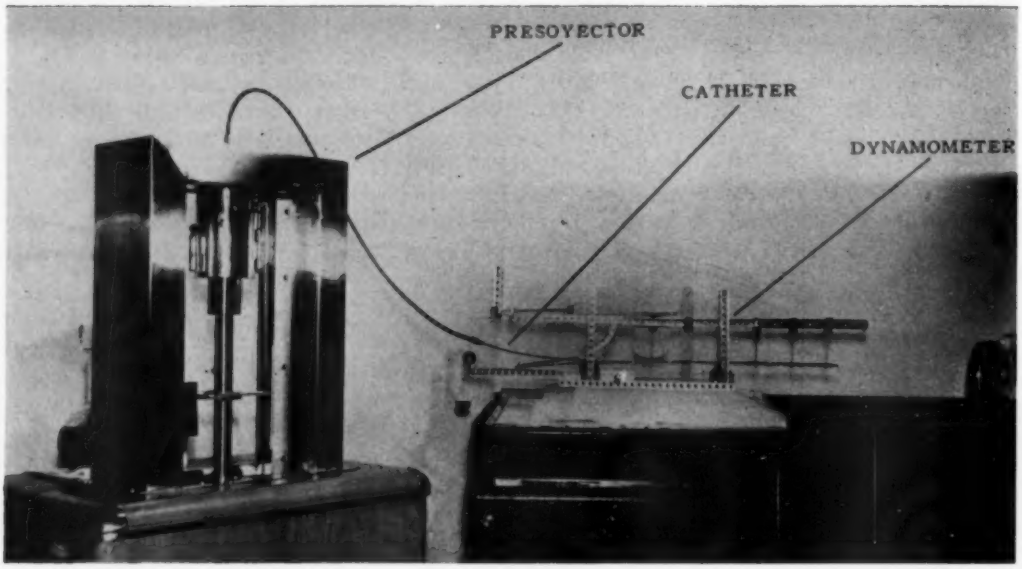


Fig. 4.—Instrumentation used in these experiments.

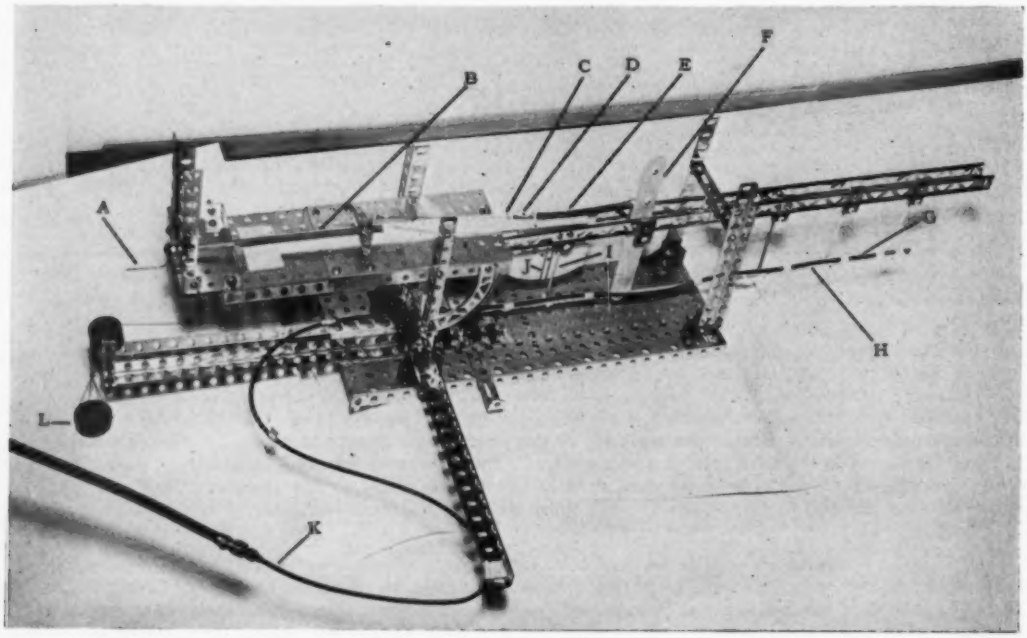


Fig. 5.—Close view of the special dynamometer for measuring the recoiling impulse of the catheters. *A*, crank mechanism which actionates screw *B*, which in turn gives the desired tension to the precalibrated spring *E*. *D*, tension indicator; *C*, scale marked in dynes; *F*, wooden lever which transmits the movements from the horizontal rod *H* to the spring *E*; *G*, catheter mounted on rod *H*. *I*, wooden rod which serves to support horizontal rod *H* and also is an indicator. *J*, vertical reference lines. *K*, catheter attached to the metallic connector. *L*, small weights used to equilibrate the system before the experiment. A weight greater than 3 grams was never necessary. It varies with the different catheter. The catheter was always mounted in the same fashion and with approximately the same bends.

In Fig. 7 the catheter with the same characteristics but with one single hole at the tip is presented when injecting at the same flow rate (27 ml. per second). Notice the great length and shape of the jet going from the tip of the catheter to the mouth of the flask in almost straight-line fashion, at least for the distance shown in the picture, 24 inches (Fig. 7, *D-D*). When injecting into the ventricles with this catheter and at this or greater flow rates, the deleterious effect of such a jet can easily be predicted. Notice the backward displacement of the system (*A-B*, Fig. 7); in this experiment a tension equivalent to 3,000 dynes had been set (the same as the one used for the catheter with lateral openings in Fig. 6); increasing the tension of the spring to 25,000 dynes brought the system back to equilibrium.

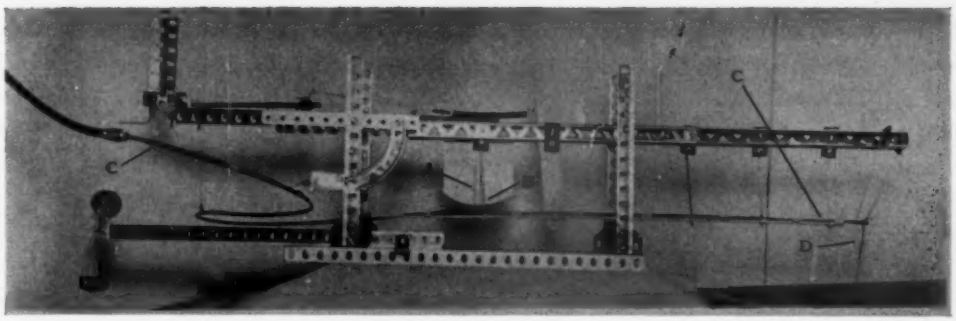


Fig. 6.—Snapshot showing an experiment in which the catheter with lateral openings was being tested, *C*. Notice that the recoiling of the catheter was not great enough to bring the system back into equilibrium. (Indicator *B* was not located between the two reference lines at *A*.) In this experiment the flow obtained was 27 ml. per second and the tension given to the spring was 3,000 dynes. Notice also the jets of fluid coming out from the lateral openings *D*. For the purpose of obtaining contrast for photography, we used water stained with T-1824.

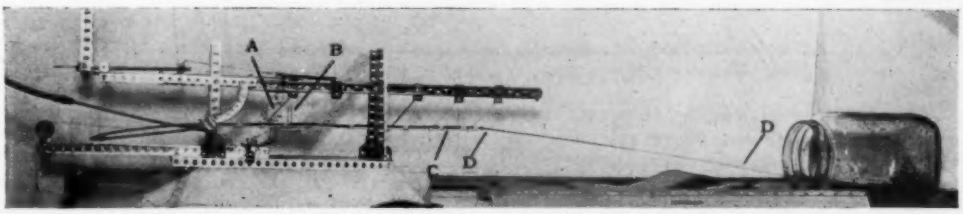


Fig. 7.—Snapshot showing an experiment in which a catheter similar to the one shown in Fig. 6, but with only one terminal hole. Size 8F, 100 cm. long. Notice that during injection there is a great backward displacement of the system to which the catheter *C* is attached; the indicator *A* is to the left of reference lines *B*. In this experiment a tension of 3,000 dynes was given to the spring prior to the injection. The injection was repeated under similar conditions except for the tension which was set at 25,000 dynes. This new setting of the tension made the indicator locate between the two vertical reference lines *B*. In both experiments the flow obtained was 27 ml. per second. Notice also the great length of the jet *D* and *D* which in these experiments traveled more than 24 inches.

## DISCUSSION

Although injections into the right ventricle have been performed successfully by using No. F9 or F10 Cournand-type cardiac catheters with a single hole at the tip, this was found dangerous because of the frequency of disturbances of the heart rhythm.*[11] It was pointed out by Jönsson and associates[11] that these disturbances were due to the mechanical effect of the jet of fluid breaking against

*Rodríguez and Sodi-Pallares[21] elicited ventricular extrasystoles in dogs by stimulating the interventricular septum with a jet of physiologic saline solution.

the ventricular wall. Our experiments suggested also that another important source of ventricular stimulation may arise from the recoil of the catheter. The catheter in recoiling would hit the opposite wall, providing this other source of stimulation to the heart. It is clear that the findings in vitro could not compare exactly with what actually happens when injecting in in vivo experiments, but the only means we have of comparing different catheters with the desired degree of accuracy is to perform experiments in vitro. For all these experiments concerning the measurement of force we used distilled water instead of radiopaque material. In Fig. 8 we present a graph in which the recoiling forces in dynes are plotted against the flows in milliliters per second, disregarding the pressures used to obtain such flows through the catheters. If we compare two catheters

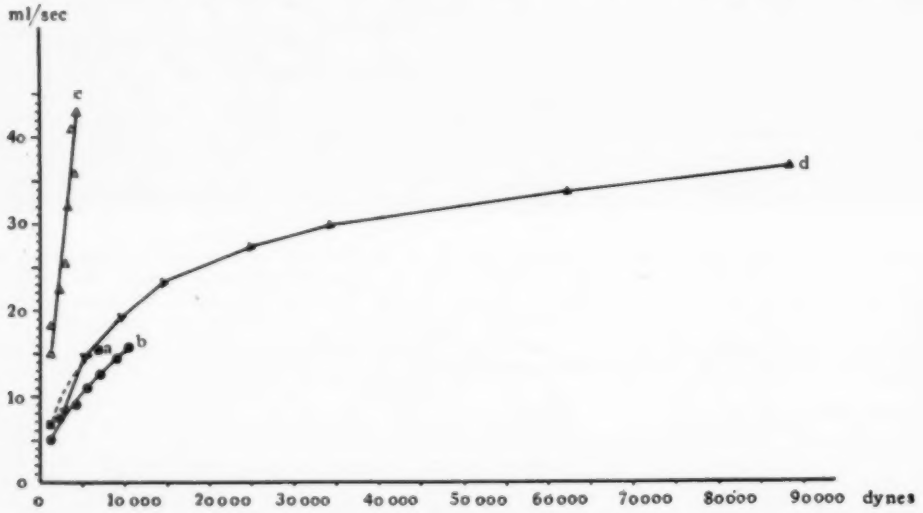


Fig. 8.—Graph correlating flow obtained through different catheters and recoil force in dynes. (*a*) Cournand cardiac catheter 8F, 100 cm. long. (*b*) Lehman Aortographic 8F, 100 cm. long. (*c*) Catheter for selective angiocardiography and aortography with 6 lateral holes, presented in this paper, 8F, 100 cm. long. (*d*) Catheter similar to (*c*) but with only one terminal hole at the tip. Notice the tremendous difference in magnitude of the recoiling force between catheter (*c*) and (*d*). In this experiment water was used as the injecting fluid.

made of the same material, having the same length and the same bore, but one with a terminal hole and the other one with the lateral openings devised by us, it can be easily appreciated that for a flow of 35 ml. per second, the catheter with a terminal hole will give a recoiling force of 78,000 dynes, whereas the one with lateral openings will give the same flow with a recoiling force of 3,200 dynes. The tremendous recoiling impulse of the catheter with one terminal hole can be easily appreciated if we realize that 78,000 dynes would be enough to lift a weight of 79 grams. The deleterious effect of such catheters should be emphasized again, especially so if great flows can be obtained with them.* On the other hand, the lateral openings in the way we arranged them could not completely avoid the recoiling force, due to the fact that the jets originating at the openings were not exactly at a 90 degree angle in respect to the catheter. (Actually this

*Warning against all catheters with one hole at the tip, especially if they produce great flows.

angle varied from approximately 78° to 98° in different jets, Fig. 6,*D*.) Jets at 90 degrees angles perhaps could have been achieved if we had used a thicker wall or smaller openings, but these would have decreased either the flow rate or the ejecting surface.

We were able, working with experimental catheters, to abolish completely the recoiling force or even to reverse it by simply varying the angles at which the openings were made with respect to the catheter, provided that the opening was small enough and the wall thick enough to really make the jets take the desired angle. This became more difficult to achieve as we made the wall of the catheter thinner. The possibility of making specially tailored catheters for each particular chamber and position of the catheter within the "organ host" (by varying the angles of the jets) was given some thought by us, but at this moment it seems impractical, since with the catheter that we have been describing, we diminished the recoiling impulse to a degree which was in the same order of magnitude as that of the elastic forces of the walls of the catheter, created by the unpredictable curvatures imposed on it as a consequence of the anatomical position of the heart chambers and great vessels, not to mention the even more unpredictable movements which constantly are taking place in a living heart.* Therefore, we present a catheter with which we can obtain a minimum recoiling force which will not interfere with selectivity, but which will permit us to have a wider bore, thinner walls, and greater ejecting surface.

Even when the recoiling force has been reduced to a point where it is negligible, we should not disregard the importance of the lineal velocity of the jets. We eliminated one of the causes of important mechanical stimulation of the heart walls, that due to the catheter itself recoiling against the walls of the heart. But we still have to analyze the danger of stimulation produced by the individual jets of radiopaque material hitting the wall opposite the openings of the catheter.† This danger can be minimized if we are able to decrease the lineal velocity of these jets. We said that in our catheter this average lineal velocity was decreased to one-tenth of that of similar catheters with one terminal hole as shown in Fig. 3. Therefore, the distribution and arrangement of the openings as described are the ones that compromise between a minimum recoiling force compatible with selectivity, a maximum flow rate, and a minimum lineal velocity of the jets of radiopaque substance. Another advantage of the lateral openings is that it facilitates the rapid mixture of blood with the radiopaque substance.[20]

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*Mechanics of the radiopaque fluid with respect to the catheter during injection has not been mentioned, but it must be kept in mind that in this technique the catheter is not straight but bent in order to conform to the anatomical position of the heart chambers; the interior surface of a bend in a catheter during injection is subjected to a pressure due in part to the actual pressure of injection which gradually diminishes towards the tip, and in part to the fact that the direction of the momentum is changed.[19] This creates tension forces in the walls of the catheter. These tensions are independent of the angle, and any curvature however small of an unsupported catheter may produce them. Since we were able to measure impulses as small as 1,500 dynes, it is felt that the resultant of all these tensional forces are below this range of magnitude.

†In the case of an open jet which is deflected from its course by a fixed, curved, and smooth surface (the internal surface of the heart walls), it may be assumed that the velocity is changed in direction only, the jet being deflected through an angle $\theta$. The resultant force exerted upon the walls would be greater as this angle approached 90°, but in the particular case of the ventricles, an open jet would exist only during very short periods of time. The normal ventricles are empty of blood only during a very short period of time during early diastole and at this time radiopaque material may still be flowing from the catheter, so that these fluids occupying the ventricles may greatly decrease velocity and, thus, the force exerted upon the walls of the heart.

It must be kept constantly in mind that the catheter with lateral openings was designed for a specific purpose and, therefore, should be used only for injecting into the chambers of the heart and great vessels or into vessels of a much greater caliber than the catheter itself. A small vessel (one with the same caliber as the catheter) would be obliterated by the catheter (a possibility applicable to all catheters), and if an injection is made, the walls of the vessel injected is subject to damage or rupture and, therefore, this possibility of damage to these walls contraindicates its use. For the sake of safety we will examine in some detail several different "organ hosts" where the injection would entail some danger. When entering the proximal aorta through the radial[22] or femoral arteries, or through any other artery, precautions must be taken not to go too far down into the proximal portion of the aorta, since the risk of obliteration of, or penetration or injection into the coronary artery exists. The standard catheter with a hole at the tip will have sufficient recoil impulse during such accidental injection that a backward displacement of the tip will occur, thus minimizing this particular risk.[23,24] This is not the case with the catheter with lateral openings. The precaution should be taken of not placing the tip any closer than within one to one and one-half inches of the semilunar valves (measurements refer to adults).

In the right side of the heart the main danger is the catheterization of the coronary sinus, due to faulty technique or to spontaneous change of location of the catheter. This happens occasionally during the time elapsing between the fluoroscopic positioning of the tip and the actual injection of the radiopaque substance. It is mandatory in this technique of selective angiocardiography not to move the patient once the catheter has been placed in the desired position.* When injection is to be made into the right ventricle, the best way of preventing this accident is to place the tip of the catheter in the pulmonary artery and then withdraw it into the ventricle while recording the pressure. An additional pressure recording just prior to injection will further detect any change in the location of the tip since it is possible that the movements of the heart, respiratory movements, and accidental or incidental movements of the patient will make the catheter drop back into the auricle and eventually enter into the coronary sinus. A very easy further check on the position of the tip (without moving the patient) in cases where it is suspected to have entered the coronary sinus is to measure the oxygen saturation of a blood sample, by means of a cuvette oximeter,† since we know that samples obtained from the coronary sinus have a low oxygen saturation. (Cuvette oximeters give an immediate saturation reading.) Accidental injections into the portion of the coronary sinus very close to the atrium may not be necessarily dangerous, but it is advisable not to do it.

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*In general, this can be achieved in three different ways: (1) Special seriographic equipment has been manufactured for the purpose of taking biplane pictures without moving the patient. (2) Recently, the electronic screen intensifier has made possible reliable cineangiocardiography. (3) In this laboratory we have provisionally adapted a Fairchild roll-film camera in order to place it under the x-ray table, and thus avoided moving the patient once the tip of the catheter has been placed into the "organ host." Once the catheter has been placed in the desired position under fluoroscopic control, the fluoroscopic tube is removed and the Farichild camera is located in its place. A hydraulic jack facilitates the positioning of the camera.

†We use the cuvette oximeter devised by Wood and Geraci.[25] Obtainable from The Waters Corporation, Rochester, Minnesota.

When the right atrium is to be injected, the tip of the catheter should be placed at the superior vena cava or at the superior portion of the atrium. Injections into the left atrium or ventricle (catheter passing through a septal defect) can be made if the position of the tip is determined by studying with the cuvette oximeter the oxygen saturation of a sample of blood withdrawn through the catheter, as well as by pressure studies. Injections at low flows into pulmonary veins can be made provided the vessels have a diameter several times greater than that of the catheter; this is true only near the atrial attachment of these veins. Injections into the pulmonary artery can be made only in the main artery and its two branches. For injecting into a smaller vessel a standard cardiac catheter with a hole at the tip must be used, particularly since, because of the small portion of the cardiac output going through that small vascular area, there is no need of injecting a great amount of radiopaque material per unit of time. Injections can be performed with a Presoyector at a low selected pressure or even with an ordinary glass syringe.[26,27]

Even if we solved most of our problems of injection with the use of the Presoyector and the special catheter described, a decrease in the total amount of radiopaque substance injected would be welcomed at any time, since it would mean a reduction of the dangers attached both to the presence of this chemical in the blood stream (and its distribution to the tissues) and to the smaller volume of the liquid injected into the "organ host." The latter would signify a lower flow through the catheter and, with it, a smaller recoil impulse and smaller lineal velocity of the jets. The total amount of radiopaque substance was fixed in the last few years at from 1 to 1.2 ml. of 70 per cent Diodrast* per kilogram of body weight. With the use of selective injections, if made rapidly enough, an excellent contrast can be obtained with smaller total amounts of radiopaque substance.[12†] Recently, Boerema and Blickman[28] published their experiments on dogs with a technique that undoubtedly will be of tremendous value when applied in connection with the high-pressure injecting machines and suitable catheters for selective angiocardiography and aortography; the advantages at least for some particular cases can easily be predicted. Their experiments consisted in raising the intrabronchial pressure for a few seconds while the injection of radiopaque substance was carried out. Since a great decrease in cardiac output is obtained, a higher concentration of radiopaque material is achieved in the "organ host." This undoubtedly will mean smaller total doses of contrast media and injections at lower flows.

The slowing down of the circulation may also be of benefit in some cases. Alterations of the normal patterns of pressures in the heart chambers may be considered sometimes as an advantage. Celis and associates[29] recently used a similar technique in human beings, by having the patient make a Valsalva maneuver during injection. With well-trained patients they were able to perform angiocardiographic studies before and after the Valsalva maneuver in fifty-eight individuals who were healthy from the circulatory point of view. Important information was obtained concerning the behavior of the circulation during these experiments which did not exactly duplicate that obtained by Boerema and Blick-

*Registered trademark.

†The same reduction in dose has been found in the case of selective injections of blue dye for the performance of dye dilution curves.[30]

man,[28] since the two experiments were not exactly the same. It can be foreseen that selective angiocardiography will take advantage of these techniques, setting decreased doses of radiopaque material and, with them, lower toxicity; by injecting lower volumes, a lower incidence of rhythm disturbances during injection may also be obtained. This will make for greater and perhaps safer selectivity.

We have recently injected 0.80 ml. of 70 per cent Urokon* Sodium per kilogram of body weight. In a case in which an injection was made into the superior vena cava, an outstanding contrast was obtained. The chambers of the heart, the pulmonary artery, and the aorta were clearly shown. This permitted the making of a diagnosis of extravascular thoracic tumor which was confirmed later at surgery. The injection of 65 ml. was made in 2.6 seconds using a pressure (actual injecting pressure) of 420 pounds per square inch, at 37° C. and through a No. 8F catheter, 100 cm. long, with lateral openings.

The newly developed radiopaque material sodium and methylglucamine diatrizoates (Urografin* in Germany and Renografin* in the U.S.A.) which is being found to be less toxic and more radiopaque than former products is, at the same time, more viscous. With our equipment we have found that this is not an insurmountable difficulty since flow rates of 31.2 ml. per second have been obtained with 76 per cent Renografin, warmed to 37° C., when injected under a pressure of 560 pounds per square inch and through a No. 8F catheter, 100 cm. long. Under the same conditions but with a No. 6F catheter, the flow rate obtained was 12.6 ml. per second.

It is believed that more highly concentrated materials could be injected with this equipment. Flow rates will be investigated with Renografin 85 per cent, which is now being prepared for experimental purposes.

## SUMMARY

1. A brief review of the techniques for selective angiocardiography was made. From these reports it was concluded that a need existed for the use of a special catheter in connection with high-pressure injecting machines.

2. A catheter devised for selective angiocardiography and aortography was presented.

3. In order to evaluate quantitatively the recoil force created at the tip of this and other catheters, a special dynamometer was devised and constructed. The results obtained by such means were reported.

4. Other physical characteristics of these and other catheters were established and reported.

5. It was concluded that the catheter described would fill the need for a selective and safe injection of radiopaque material when used in connection with the Presoyector or other safe mechanical injector.

6. Since the catheter was devised for specific uses, the dangers involved in its use for other purposes were mentioned. Warning was given also against the use of catheters with one terminal hole at the tip (especially if great flows could be obtained with them) for the purpose of high-pressure injections of radiopaque substances.

7. The application of recent techniques using increase in intrabronchial pressure to enhance the contrast of angiocardiographic plates was briefly discussed.

*Registered trademark.

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# P VECTOR LOOP IN HEALTH AND DISEASE AS STUDIED BY THE TECHNIQUE OF ELECTRICAL DISSECTION OF THE VECTORCARDIOGRAM (DIFFERENTIAL VECTORCARDIOGRAPHY)

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ALTHOUGH there have been many reports on vectorcardiography, we are not aware of any systematic study of the P vector loop. The main reason is the technical difficulty of obtaining unblemished records. The P loop is relatively small and merges with the initial and the terminal portions of the QRS and ST-T loops. With ordinary methods of photographic recording, the details of the P loops are obscured.[1] Although parts of the P loop occasionally stand out by chance, its origin, termination, and direction of inscription are not usually apparent.[2] Frequently, the ST-T loop or some abnormal part of the QRS loops is regarded as the P loop, or vice versa. The developing method of Milovanovich[3] is helpful in determining the direction of the QRS loop and in identifying the ST-T and P loops, but it distorts the P loop, thereby precluding its detailed analysis. Motion picture photography is not practical. The technique of deriving planar projections of the PsĚ loops from the contour of the heart on autopsy[4] is laborious and based upon doubtful assumptions. The recent development of differential vectorcardiography[5,6] has made it possible to study the fine details of the component loops of the vectorcardiogram. In the present report, the characteristics of P loops of the normal heart, and of right and left atrial hypertrophy will be presented.

## MATERIAL AND METHODS

*Criteria for Selection of Cases.*—The well-established criteria of the normal P wave of the normal heart have been followed in this study.[7,8] The contour of the P wave is generally smooth. The normal duration varies from 0.07 to 0.12 second, usually being less than 0.10 second; the mean is 0.09 second and the standard deviation 0.015 second. The amplitude of the P wave in the limb leads ranges from 0.5 to 2.5 mm., with an average value of 1.3 to 1.4 mm. in Lead II.[7] In the Wilson chest leads, the P wave is usually biphasic or negative in $V_1$ and $V_2$, and positive in the left chest leads.[8]

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Two electrocardiographic patterns of left atrial hypertrophy have been studied (Figs. 5, 6, 12, 14). Type I is the classical type designated as mitrale P, and is found most commonly in mitral stenosis. The duration of the P wave usually exceeds 0.10 second, and a double summit is distinct in Leads I and II, and in left precordial leads. The amplitude of the P wave usually exceeds 0.1 mv. (1 mm.) in Lead I. P is upright, broad, and notched in Lead I or II, and upright or inverted, notched, and diphasic in Lead III. $V_1$ or $V_2$ shows diphasic (plus-minus) or negative P wave, and the left precordial leads have broad, upright, notched P waves. Type II left atrial hypertrophy is characterized by a tall P wave, especially in Leads II and III, ranging from 0.1 to 0.4 mv. (average 0.23) in amplitude, and usually exceeding 0.10 second in duration. In the frontal plane (standard and unipolar limb leads) Type II resembles pulmonale P waves, although the latter tend to be more peaked. However, in the horizontal plane, Type II has either diphasic or negative P waves in $V_1$ or $V_2$, and positive P waves in the left precordial leads.

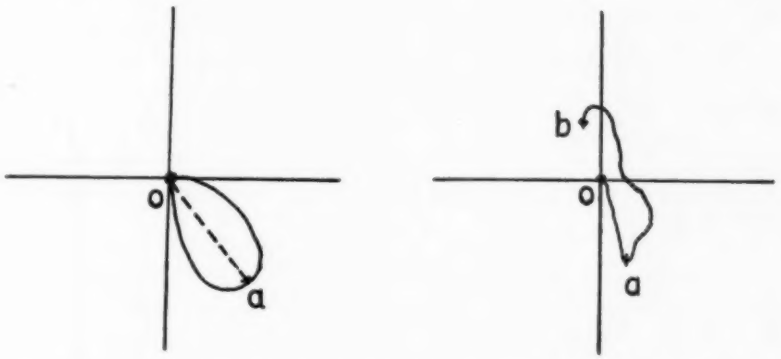


**Fig. 1.—Measurement of maximal vector (*oa*) and maximal length (*ba*) of P loop. *O* indicates the origin, *b* and *a* the termination of the P loop. *ob* represents the Ta displacement of the electrocardiogram.**

The cases with right atrial hypertrophy presented the classical pattern of pulmonale P, characterized by a tall and peaked form in Leads II, III, and aVF, with an average amplitude of 0.23 mv.; in the horizontal plane, the P wave is usually upright and peaked in $V_1$ and $V_2$, and is taller in $V_1$ and $V_2$ than in $V_4$, $V_5$, or $V_6$. Some cases of right atrial hypertrophy showed diphasic P waves in $V_1$ or $V_2$, resembling the Type II of the left atrial hypertrophy. However, in the latter the negative deflection in $V_1$ or $V_2$ tended to be broader.

Except for a few cases, to be discussed later, the electrocardiographic criteria of atrial hypertrophy were constant features in serial records of the same patient. In order to determine the site of atrial hypertrophy, we considered the whole clinical picture of each case, including right heart catheterization, x-ray, and other laboratory data, as well as operative notes.

The dissected P vector loops of 81 cases were studied: 16 normal, healthy house officers or medical students; 15 cases with practically normal P waves; 23 cases with left atrial hypertrophy (10 Type I, and 13 Type II); 3 cases with bor-

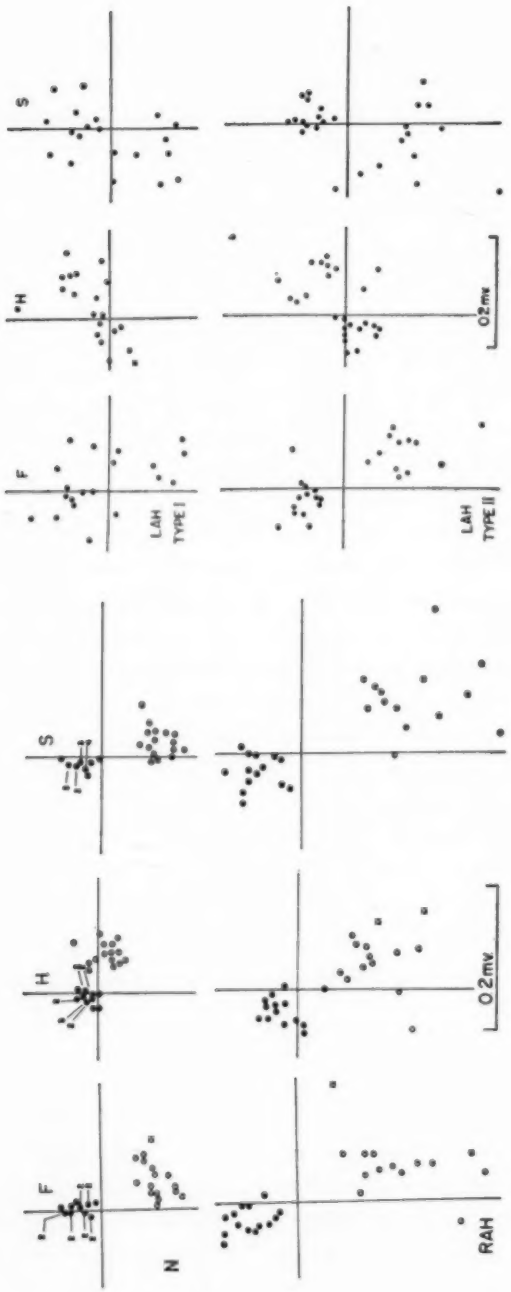


Fig. 2.—Location of the termini of the maximal P vectors (open circles) and Ta junction vector (solid circles) in the frontal, sagittal, and horizontal planes in: *A*, Normal (*N*) and right atrial hypertrophy (*RAH*); *B*, Types I and II left atrial hypertrophy (*LAH*). (Discussed in text.)

derline left atrial hypertrophy; 1 case with probable right atrial hypertrophy; 14 cases with right atrial hypertrophy; 5 cases with combined atrial hypertrophy; and 4 cases with ectopic origin of the P wave. Observations on the morphology of the P loop have been made in an additional 250 cases where dissection was not performed. However, to avoid errors which are intrinsic in the analysis of "undissected" PQRST loops recorded in the usual fashion, these cases have not been included in this study.

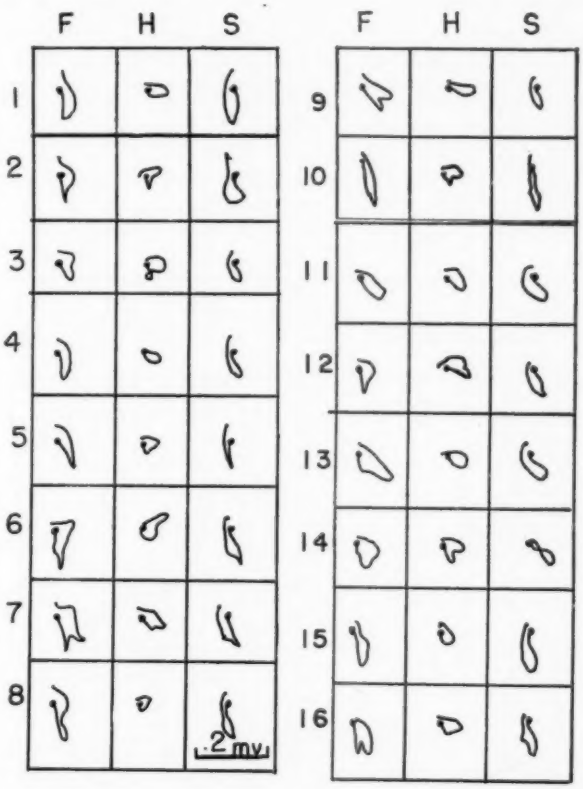


Fig. 3.—Tracings of P vector loops of 16 normal persons. *F*, *H*, and *S* indicate frontal, horizontal, and sagittal planes. The origin is indicated by the heavy dot. (Discussed in text.)

The normal subjects were male medical students or house officers who ranged from 25 to 35 years of age and showed no evidence of heart disease by either history, physical examination, or conventional electrocardiography.

The group with "practically normal" P waves had various kinds of heart disease and *normal P waves*. Nine had acute myocardial infarcts, and the remainder had pericarditis, myocarditis, or left ventricular hypertrophy. Some manifested evidence of left heart failure. The purpose of including this group was to determine whether the vectorcardiogram yielded information not obvious in the electrocardiogram.

All of the patients with Type I left atrial hypertrophy had mitral stenosis, confirmed by clinical studies, catheterization and/or operation. Two patients

also had aortic stenosis. Patients with clinical evidence of associated right atrial hypertrophy and strain (secondary to mitral stenosis) were excluded from this group.* As will be mentioned later, on the basis of vectorcardiographic changes, Cases 2, 7 with Type I, and Cases 1, 2, 3, 6, 12 with Type II left atrial hypertrophy, probably had some degree of right atrial hypertrophy.

Seven of the patients with Type II left atrial hypertrophy had arteriosclerotic and/or hypertensive cardiovascular disease with evidence of left heart failure, but not mitral deformity. The remainder (6 cases) had mitral stenosis.

Fig. 4.—Tracings of P vector loops of 15 patients with heart disease and normal P waves. (Discussed in text.)

Eleven of the patients with right atrial hypertrophy had chronic cor pulmonale secondary to pulmonary tuberculosis, bronchiectasis, bronchial asthma, berylliosis, or pulmonary fibrosis. Two patients had congenital heart disease, with clinical evidence of right failure (interatrial septal defect, patent ductus arteriosus with reversal of flow). One remarkable case (Case 6) developed severe right heart failure secondary to a surgically produced aorta-coronary sinus anastomosis.[9] Cardiac catheterization data indicated a shunt flow of 2 liters per minute. The anastomosis was closed surgically and evidence of right heart failure subsided within a week.

*It is probable that in many cases of atrial hypertrophy both atria undergo hypertrophy. However, th e predominant hypertrophy is in the atrium with the greater work load.

*Vectorcardiographic Technique.*—A modification of Duchosal's reference system was employed.[10] Details of our apparatus and electrical dissection have been presented elsewhere.[6] Briefly, the dual beams of a Dumont cathode-ray oscilloscope were intensified only during the inscription of the P loop. Simultaneously, a direct-writing electrocardiogram was marked during the period of unblanking (intensification). In the latter part of this study, the P wave was inscribed by one beam of the cathode-ray oscilloscope and photographed simultaneously with the P loop. The direction of the movement of the beam was noted by direct observation. In a few cases partial dissection of the P loop was necessary to determine the direction of rotation. Photographs were taken by a polaroid Land camera. Generally, the sensitivity was adjusted so that an input of 1 mv. produced a 6-inch deflection on the cathode-ray oscilloscope. Thus, with

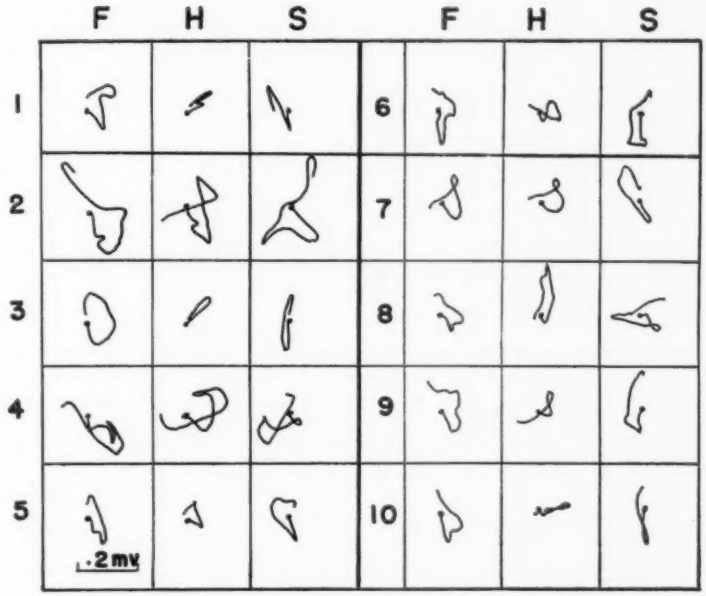


Fig. 5.—Tracings of P vector loops of 10 patients with Type I left atrial hypertrophy (mitrale P waves). (Discussed in text.)

each millivolt input, the beam moved downward and to the observer's right, at an angle of 45 degrees and with a magnitude of 6 $\sqrt{2}$ (8.5) inches. Because of the camera reduction ratio (2.25), a deflection of 1 inch on the cathode-ray oscilloscope produced an image of 11.3 mm. on the developed Land film. With the 6-inch standardization and the camera used, each millimeter deflection on the developed film was equal to an input of 0.0148 mv. The magnitude of the maximum vector of each P loop was determined and the angle of the maximum vector in the frontal, horizontal, and sagittal planes was measured. In a few cases with two large and widely divergent vectors the angle of each vector was measured. In a few loops with two vectors of about the same magnitude and direction the mean value was determined.

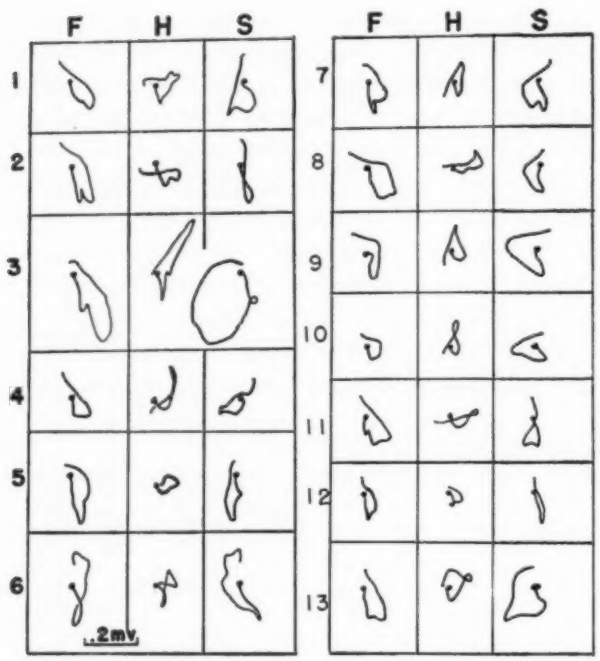


Fig. 6.—Tracings of P vector loops of 13 patients with Type II left atrial hypertrophy. (Discussed in text.)

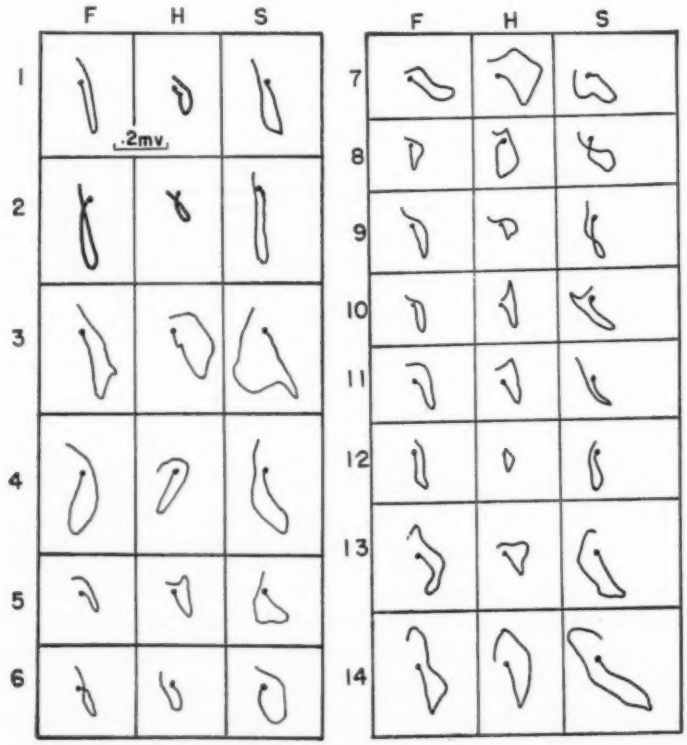


Fig. 7.—Tracings of P vector loops of 14 patients with right atrial hypertrophy. (Discussed in text.)

Since in pathologic states the P loops are often open widely, two measurements were made: (1) maximum vector (M.V.) which is a straight line drawn from the origin of the loop to its most distant point; and (2) maximum length (M.L.) which includes the open end of the loop, corresponding to Ta displacement of the electrocardiogram (Fig. 1). The difference between the maximum vector and maximum length is a measure of the opening of the loop, i.e., Ta displacement.

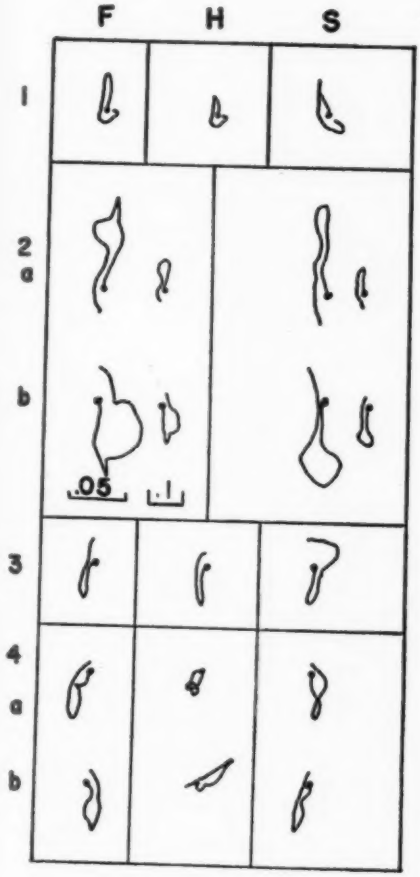


*Fig. 8.*

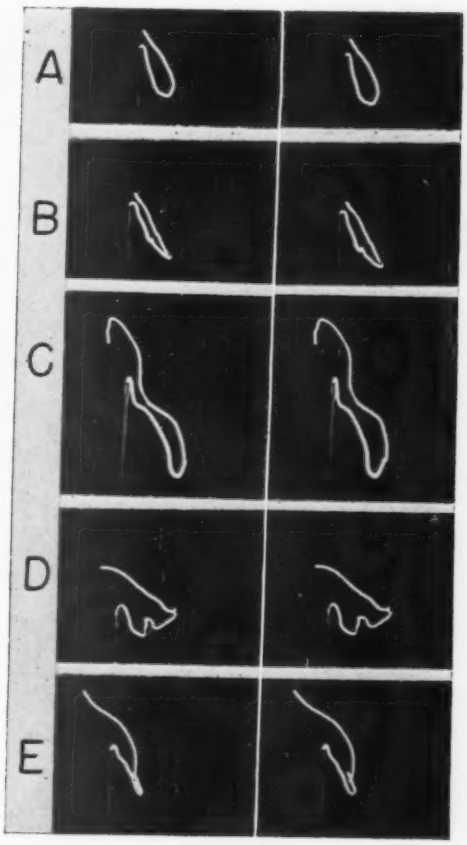


*Fig. 9.*

Fig. 8.—Tracings of P vector loops of 4 patients with ectopic rhythms. *2b* and *4b* are P loops originating from the S-A node. (Discussed in text.)

Fig. 9.—Stereophotographs of P vector loops of: *A*, Normal subject; *B*, Normal subject (vectorcardiogram shown in Fig. 10); *C*, right atrial hypertrophy (vectorcardiogram shown in Fig. 17); *D*, Type I, left atrial hypertrophy (mitrale P wave); and *E*, Type II left atrial hypertrophy (vectorcardiogram shown in top row, Fig. 13).

The standard bipolar, augmented unipolar limb lead, and six chest lead electrocardiograms were taken immediately before or after the vectorcardiograms were recorded. A wire model of the PsĒ loop was constructed in each case from the frontal, horizontal, and sagittal vectorcardiograms in order to elucidate the three dimensional aspects.

## RESULTS

The results listed in Table I include the magnitude of the maximum vector, the maximum length of each P loop, and the angle of the maximum vector with the X axis in the frontal and horizontal planes, and with the Y axis in the sagittal plane. In the frontal plane, the angles were positive if below and negative if above the X axis. In the sagittal plane, the angles were measured from the Y axis, being considered positive if located to the right side (of the observer), which corresponded to the anterior part of the patient, and negative if located to the left (posterior). The location of the maximum vectors in the various etiologic groups is presented in Fig. 2. Drawings made from tracings of the vectorcardiograms are illustrated in Figs. 3 through 8.

TABLE I. ANALYSIS OF P LOOPS OF 81 PATIENTS

| DIAGNOSIS | FRONTAL PLANE | | | HORIZONTAL PLANE | | | SAGITTAL PLANE | | |
|---|---|---|---|---|---|---|---|---|---|
| NUMBER OF CASES | ANGLE (DEG.) (S.D.) | M.V. mv. (S.D.) | M.L. mv. (S.D.) | ANGLE (DEG.) (S.D.) | M.V. mv. (S.D.) | M.L. mv. (S.D.) | ANGLE (DEG.) (S.D.) | M.V. mv. (S.D.) | M.L. mv. (S.D.) |
| Normal (16) | 70.6 (15.6) | 0.095 0.022 | 0.113 0.018 | 19.0 (17.9) | 0.053 0.016 | 0.053 0.016 | 17.8 (13.5) | 0.092 0.018 | 0.110 0.021 |
| Practically Normal (15) | 59.1 (18.01) | 0.090 0.028 | 0.118 0.041 | 4.7 (37.4) | 0.065 0.021 | 0.075 0.021 | —4.7 (25.02) | 0.084 0.028 | 0.107 0.028 |
| Left atrial Hypertrophy Type I (10) | 33.6 (28.05) | 0.111 0.020 | 0.179 0.062 | —43.0 (23.8) | 0.098 0.028 | 0.126 0.037 | —66.8 (19.5) | 0.107 0.024 | 0.170 0.047 |
| Left atrial Hypertrophy Type II (13) | 60.0 (21.6) | 0.123 0.029 | 0.185 0.041 | —13.4 (13.1) | 0.101 0.041 | 0.121 0.043 | —22.6 (10.9) | 0.127 0.036 | 0.182 0.038 |
| Borderline Left atrial Hypertrophy (3) | 58.3 (8.05) | 0.118 0.032 | 0.148 0.053 | —44.0 (26.1) | 0.114 0.007 | 0.129 0.139 | —34.7 (29.4) | 0.114 0.025 | 0.148 0.043 |
| Right atrial Hypertrophy (14) | 74.0 (18.4) | 0.161 0.053 | 0.221 0.061 | 70.1 (20.9) | 0.124 0.046 | 0.169 0.065 | 28.1 (14.2) | 0.182 0.058 | 0.246 0.071 |

*Normal P Loop.*—(Fig. 3.) The following features of the normal spatial P vector loops of healthy persons were noted: (1) *Magnitude* was small; the magnitude of the maximum vector ranged from 4 mm. to 8 mm. in the frontal plane, with an average value of 6.4 mm. (0.095 mv.); from 2 to 5 mm. (average 3.6 mm., 0.053 mv.), in the horizontal plane; and from 4 to 8 mm. (average 6.25 mm., 0.092 mv.), in the sagittal plane. (2) *Configuration* of the loops showed them to be closed in the horizontal plane with equal maximum vectors and maximum lengths, but slightly open in the frontal and sagittal planes. The difference between the average maximum vector and maximum lengths was 1.2 mm. (0.018 mv.). The termini of the P loops corresponding to the Ta junction were clus-

tered around minus 90 degrees in the frontal and sagittal planes, approximately 150 degrees from the mean P vector. The loops were comparatively smooth, except in 3 cases (Nos. 7, 9, 16) which showed two maximum vectors of about the same magnitude. (3) *Direction* of the maximum vector was vertical inferiorly

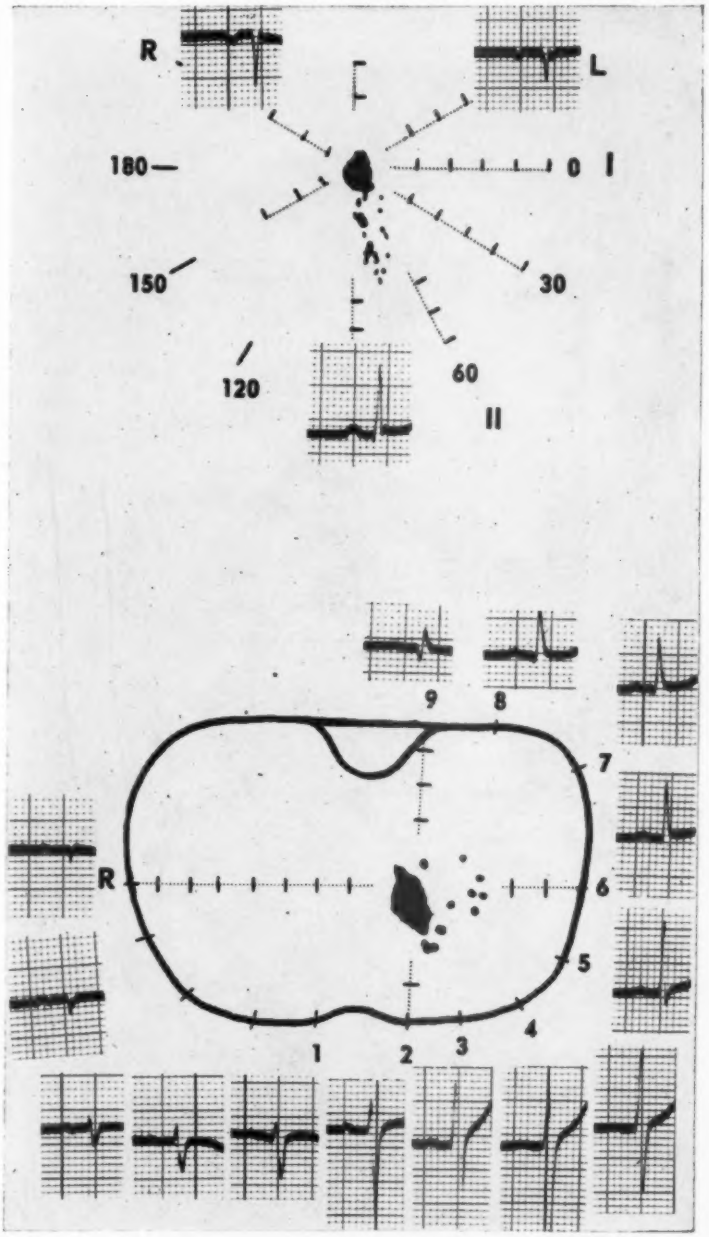


**Fig. 10.—P loops of normal subject in frontal and horizontal planes (Case 10). Bifid P waves are shown in the limb leads, especially $aV_F$, and in the chest leads $V_3$, $V_4$, $V_5$. The loop is closed. The first portion is directed inferiorly and anteriorly; the second portion inferiorly and posteriorly to the left. The inscription in both planes is counterclockwise. The cathode-ray oscilloscope beam is interrupted 200 times each second in the frontal plane and 100 times in the horizontal plane. 1 mm. deflection in the print is equivalent to 0.0019 mv. in the frontal plane, and 0.0037 mv. in the horizontal plane.**

(70.6 degrees), and slightly anteriorly (19 degrees). In the frontal plane the range of the maximum vector was between 35 to 92 degrees (average 70.6, standard deviation 15.6); between minus 10 degrees to plus 46 degrees (average 19.0, standard deviation 17.9) in the horizontal plane; and between minus 8 degrees to plus 47 degrees (average 17.8, standard deviation 13.5) in the sagittal plane.

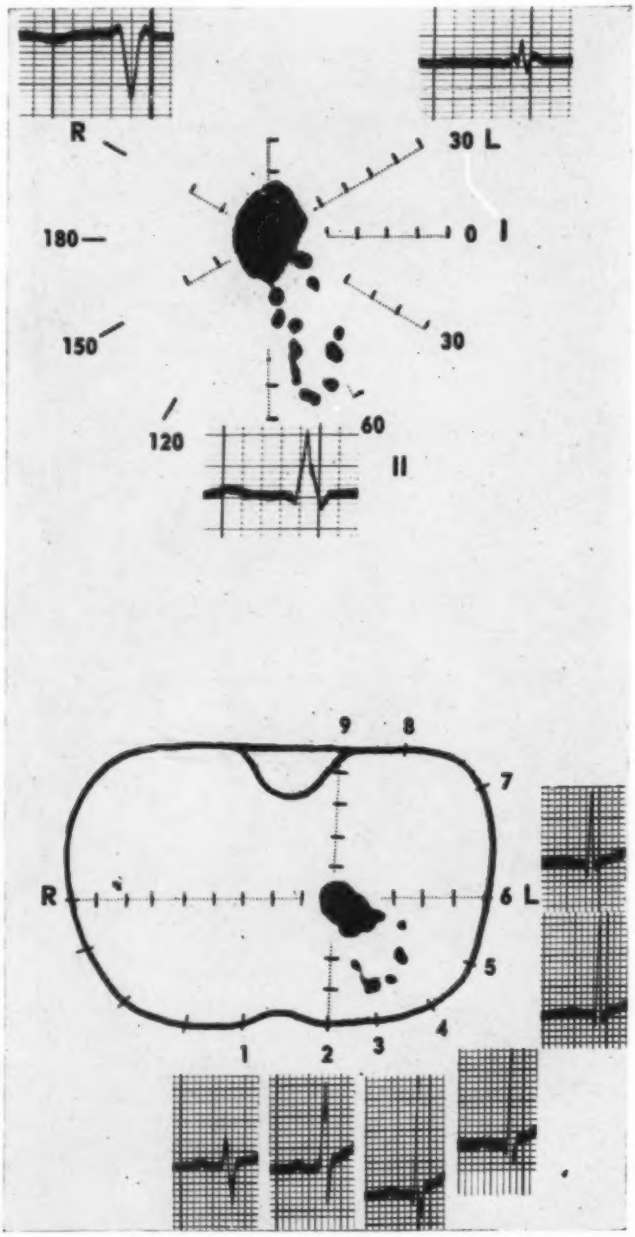


Fig. 11.—P vector loops and electrocardiogram of a normal subject (Case 15) showing counterclockwise rotation in the frontal and horizontal planes. The standardization is 0.003 mv./mm. The cathode-ray oscilloscope beam is interrupted 200 times each second in the frontal and 100 times in the horizontal planes.

(4) *Inscription* of the normal P vector loop was much more constant than that of the QRS loop. The normal P loop showed counterclockwise rotation in the frontal and horizontal planes and clockwise rotation in the sagittal plane. There were no exceptions other than in P loops originating from ectopic foci. Similar characteristics of the normal P loop in the frontal and sagittal planes were noted

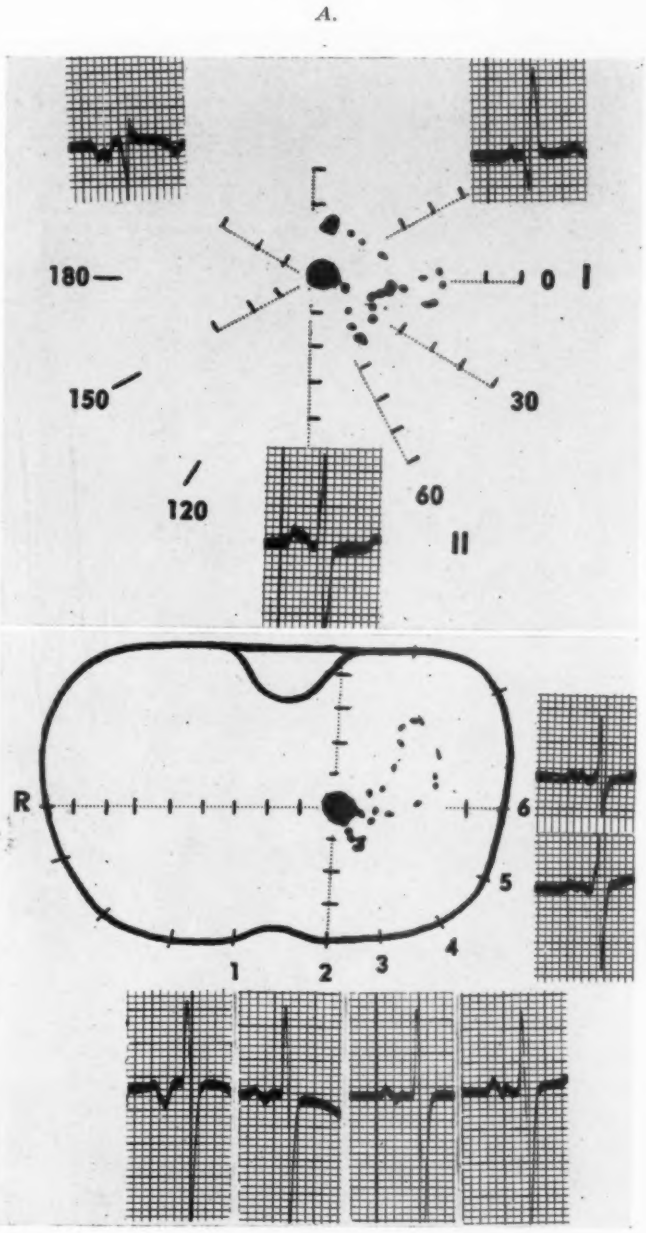


**Fig. 12.—Relationship of electrocardiogram to P vector loops of patients with Type I left atrial hypertrophy. *A*, frontal plane; *B*, horizontal plane.**

by Fowler and Dorney.[2] However, failure of the P loop to return to the isoelectric point was not observed in their study.

The three dimensional wire models of the spatial P loop revealed that the loop did not lie in one plane (Fig. 9). The plane of the first part of the loop was

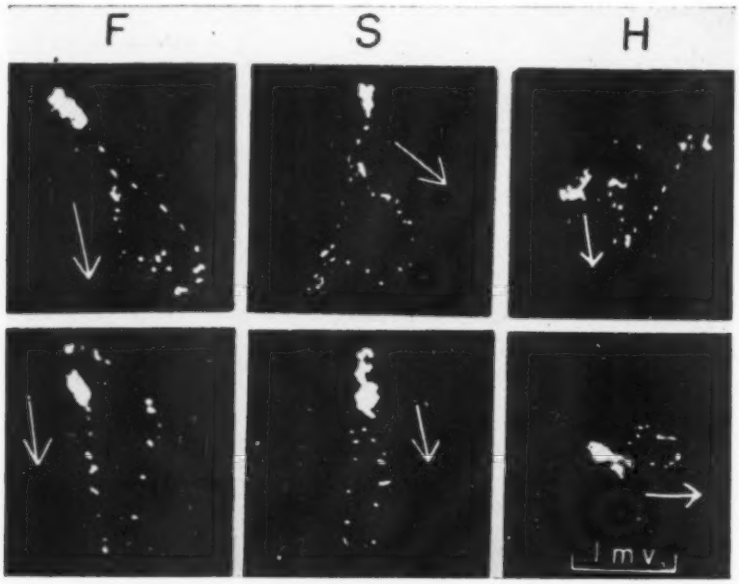


Fig. 13.—P vector loops of Cases 1 and 5 with Type II left atrial hypertrophy. (Discussed in text.)

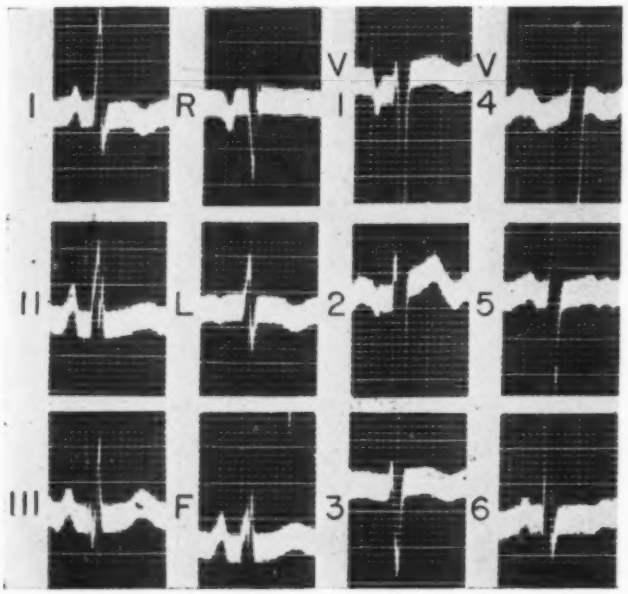


Fig. 14.—Electrocardiogram showing peaked P waves of Type II left atrial hypertrophy (Case 1) (Fig. 13, upper row). The patient had severe hypertension, blood pressure 240/178, and left heart failure.

almost perpendicular to the frontal plane of the body. The plane of the second part of the loop formed an angle of approximately 60 degrees with the first part, was directed to the left, and was almost parallel to the frontal plane. The relationship of the plane of the first and second parts of the P loop assumed a greater importance in hypertrophy. In right atrial hypertrophy the first part of the loop became larger, relatively and absolutely; and in left atrial hypertrophy the second part of the loop predominated. In combined hypertrophy, both portions were enlarged.

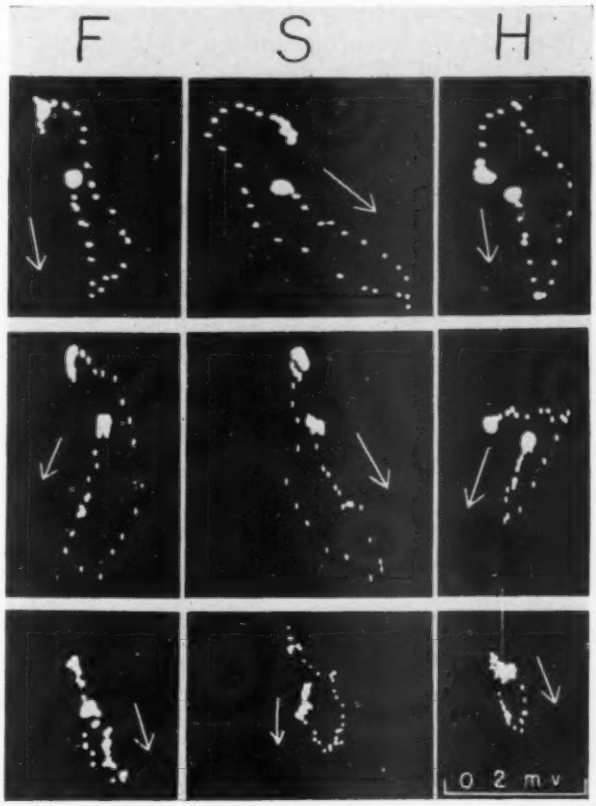


**Fig. 15.—P vector loops of right atrial hypertrophy (Cases 14, 4, 6) showing open loops with maximal vector directed anteriorly and downward. In Case 6 (bottom row) the direction of inscription was unusual, being counterclockwise in the frontal and horizontal planes and clockwise in the sagittal plane. (Discussed in the text.)**

*"Practically" Normal.*—(Fig. 4.) The vectorcardiographic features of the group with "practically" normal P waves were similar to those of the normal P loop in the healthy group. In five cases (Nos. 2, 3, 4, 7, and 8) the maximal vectors were directed more to the left and posteriorly, outside of the normal range. Case 3 had rheumatic aortic insufficiency. Cases 2, 4, 7, and 8 had acute myocardial infarction. Case 12 was directed more anteriorly than was the normal P group. The contour of the loops was smooth except in Cases 4, 7, 8, and 9. This group showed only slight or no opening of the P loop. The difference between the average maximum vector and the maximum length was 0.028, 0.010, and 0.023 mv. in the frontal, horizontal, and sagittal planes, respectively.

*Left Atrial Hypertrophy.*—(Figs. 5, 6, 9, 12, 13, 14.) The P loops differed conspicuously from the normal in magnitude, configuration, and direction. The maximum vectors of Type I (mitrale P) ranged in the frontal plane from minus 70 degrees to plus 84 degrees (average 33.6) in direction; and from 5 to 10 mm. (average 7.5 mm., 0.111 mv.) in magnitude. In the horizontal plane the maximum vector ranged from 60 to minus 85 degrees (average minus 43 degrees); Cases 2 and 7 had two large vectors; the first was directed vertically and anteriorly. The second vectors of these two cases, and the maximum vectors of the remainder, were located between minus 11 and 85 degrees. The average magnitude was 6.6 mm., 0.098 mv., with a range of 4 to 10 mm.

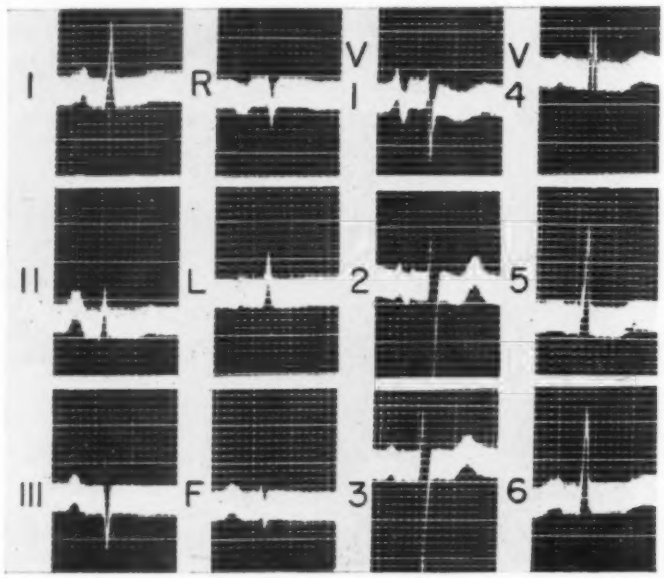


**Fig. 16.—Electrocardiogram of patient with right atrial dilatation and hypertrophy (Case 6). P vector loops are shown in bottom row of Fig. 15.**

In the sagittal plane maximum vectors ranged from plus 37 to minus 175 degrees, and from 5 to 10 mm. (average 7.2 mm., 0.107 mv.). In the frontal plane the maximum vectors of Type II ranged from plus 43 to plus 86 degrees, with the exception of Case 6 whose second large vector formed an angle of minus 80 degrees. The magnitude ranged from 4 mm. to 12 mm. (average 8.3 mm., 0.123 mv.). In the horizontal plane the maximum vectors ranged from plus 75 to minus 82 degrees. (The range in the cases with one maximum vector and of the second vector in the cases with two vectors was minus 82 to plus 40, with an average of minus 38.8 degrees.) In the sagittal plane the maximum vector ranged from plus 25 to minus 110 degrees (average minus 22.6 degrees), and from 6 to 15 mm. (average 8.6 mm., 0.127 mv.).

The P loops of the two types of left atrial hypertrophy did not differ significantly. In both types there were cases of two major vectors (Type I, Cases 1, 2, 7; Type II, Cases 1, 2, 3, 6, 7). The first vector was directed anteriorly about 70 degrees in the horizontal plane and 25 degrees in the sagittal plane.

The second major vector was directed posteriorly and downward to the left in Type II, and posteriorly and upward to the left in Type I. As will be mentioned later, it was likely that the first vector represented some degree of right atrial hypertrophy.

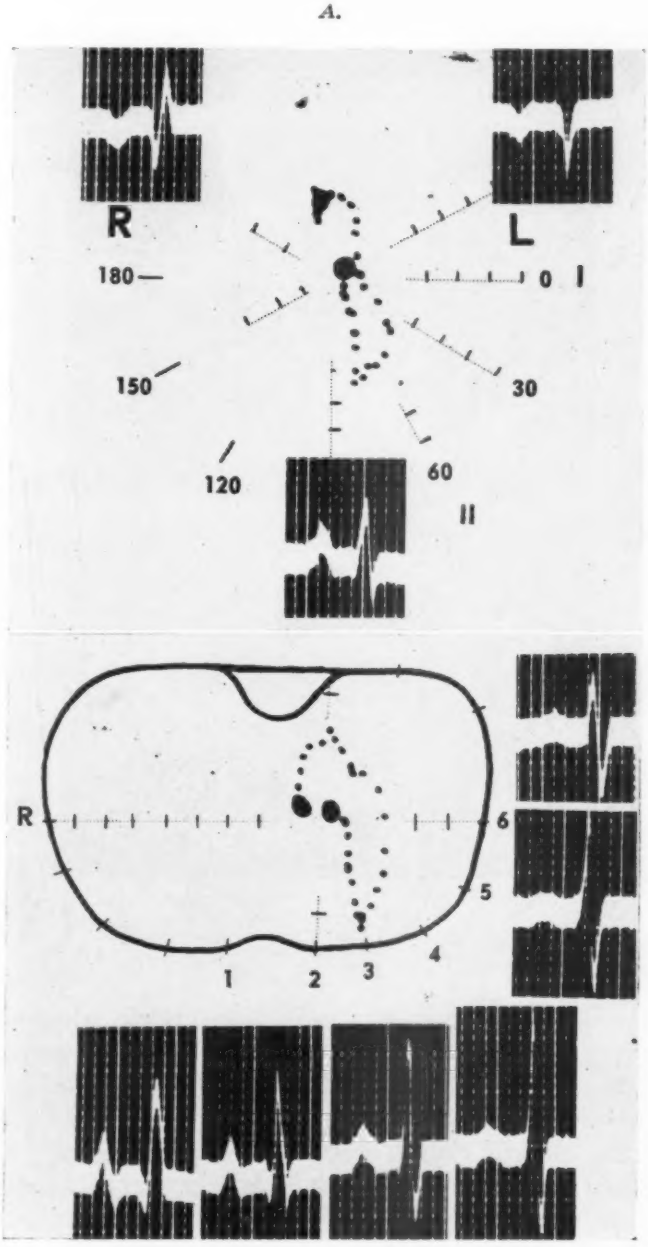


Fig. 17.—Electrocardiogram and vectorcardiogram of frontal (*a*) and horizontal (*b*) planes of patient with right atrial hypertrophy, secondary to primary pulmonary hypertension.

The vector loops of two types of left atrial hypertrophy differed in contour from each other, and differed from the normal, also (Fig. 9). The Type I (mitrale) P loop was more bizarre, especially, being bifid in the frontal plane to produce broad notched P waves in the limb leads (Figs. 9, 12). The terminal part of the loop was directed more leftward and upward than in Type II.

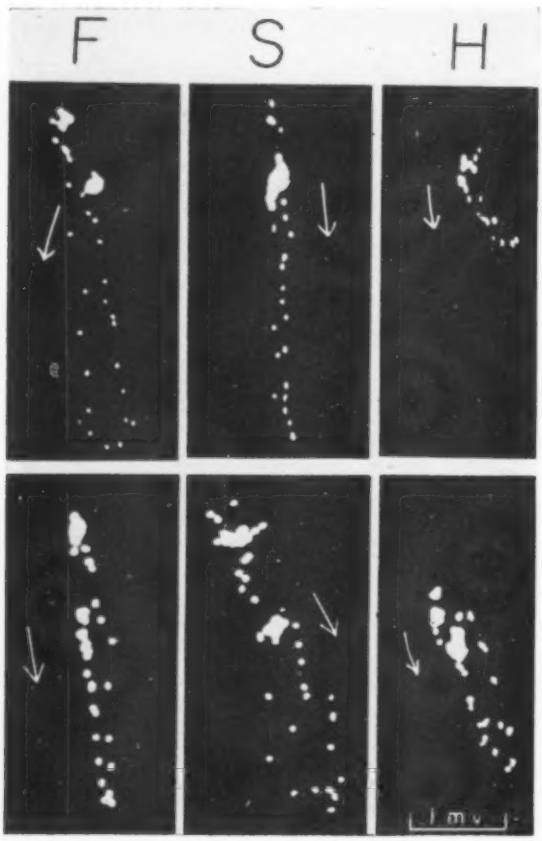


Fig. 18.—P vector loops of right atrial hypertrophy showing unusually narrow P loops (Case 2, upper row; Case 1, lower row).

Type II loops were narrow in the frontal plane, accounting for the taller P waves. The first portion of the P loop in Type II was almost perpendicular to the frontal and, hence, did not produce the first part of the double notch so manifest in Type I. In the horizontal plane in both types the maximum vector (or terminal maximum vector in cases with two vectors) was directed more posteriorly to the left than in normal cases. The principal direction of inscription was counterclockwise in the frontal and horizontal planes, and clockwise in the sagittal. In the frontal plane, the only exceptions were cases of Type I (Cases 4 and 7), and Type II (Case 6) which showed both clockwise and counterclockwise rotation. In the horizontal plane, the rotation was totally counterclockwise, except in cases with a figure-of-eight contour (Type I, Cases 2, 4, 6, 7, 9, 10; Type II, Cases 4, 6, 10, 11, 12, 13) where the terminal part was clockwise. The rotation

was clockwise in the sagittal plane in all but 7 cases. The latter had a figure-of-eight contour where both clockwise and counterclockwise rotations were present.

The three-dimensional wire models clarify the fact that the enlarged area of the terminal portion of the loop was directed posteriorly to the left, and that its plane was so rotated backward as to be almost perpendicular to the frontal plane —unlike in the normal cases where it was almost parallel.

The P loops in patients having left atrial hypertrophy were more conspicuously open than those in the normal patients. The difference between the maximum vector and maximum length was similar in both types of left atrial hypertrophy, approximately 0.065 mv. in the frontal and sagittal planes, and 0.02 mv. in the horizontal plane. The termini of the P loops were clustered around minus 120 degrees in the frontal plane and plus 145 degrees in the sagittal plane. In other words, Ta was directed anteriorly, and towards the right to the right upper octant, representing a shift of about 30 degrees from the normal position.

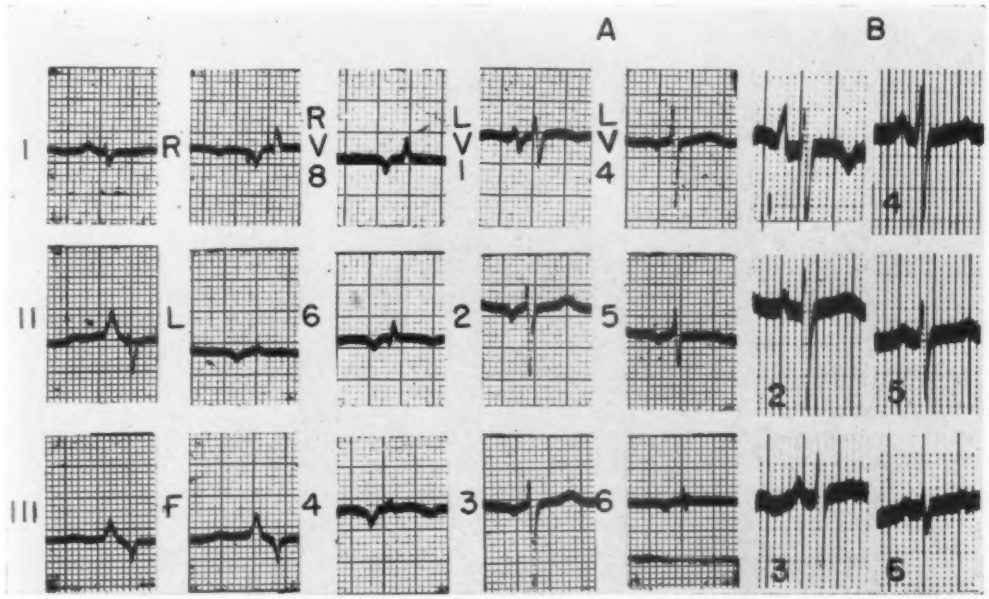


**Fig. 19.—Electrocardiogram of patient with right atrial hypertrophy showing unusually tall P waves. P vector loops are shown in bottom row of Fig. 18. The chest leads (*A*) show unusual negative P waves in $V_2$ to $V_6$ which are probably positional. A record taken the next day (*B*) showed P waves unchanged in limb leads but upright P waves in the chest leads. The P wave was unusually tall in $V_1$ in this and other records. (Discussed in text.)**

In summary, the vectorcardiographic features of the P loop of left atrial hypertrophy included an increase of magnitude, and a bizarre-shaped configuration with bifid open loops often forming figures-of-eight. The direction of the maximum vector was posterior and upward in Type I, and posterior and inferior in Type II, and in both, more to the left than the normal P maximum vector. The terminal part of the P loop was enlarged and formed the maximum vector.

*Right Atrial Hypertrophy.*—(Figs. 7, 9, 15, 16, 17, 18, 19.) The spatial P loops of right atrial hypertrophy were distinctly different (Figs. 15, 18). (1) *Magnitude.* The maximum vector ranged from 6 to 17 mm. (average 10.9 mm., 0.16 mv.) in the frontal plane; from 5 to 16 mm. (average 8.4 mm., 0.124 mv.) in the horizontal plane; and from 8 to 19 mm. (average 12.3 mm., 0.182 mv.) in the sagittal plane. The P loop of patients with right atrial hypertrophy was significantly larger than that of normal persons and of patients with left artrial hypertrophy (Table I). (2) *Configuration.* The loops had a relatively smooth contour and were open in all planes (Figs. 15, 17, 18). The magnitude of the loop opening was indicated by the difference between the maximal vector and maximal length, i.e., 0.06 mv. in the frontal and sagittal planes, and 0.045 mv. in the horizontal plane. The termini of the P loops corresponding to the Ta junction were clustered around minus 120 degrees in the frontal plane, minus 120 degrees in the horizontal plane, and minus 155 degrees in the sagittal plane (Fig. 2).

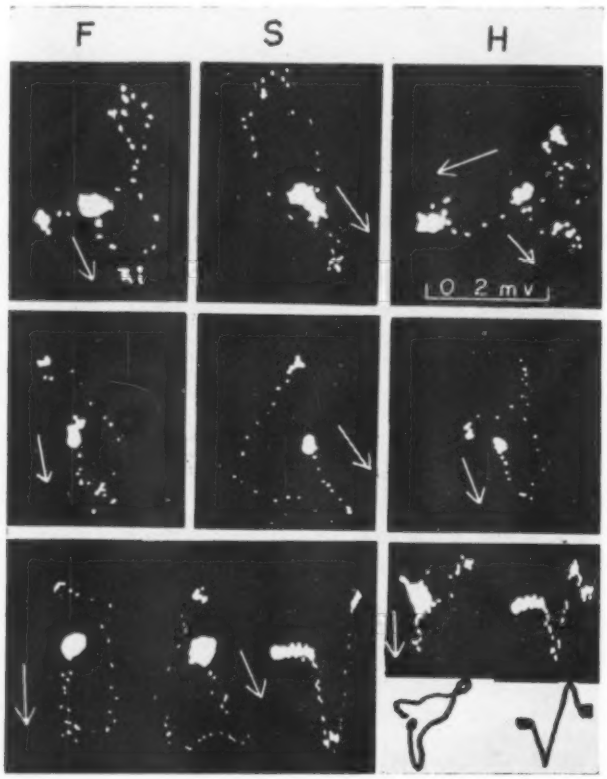


**Fig. 20.—P vector loops of 3 patients with combined atrial hypertrophy. The loops are open. The initial large early vector forces are directed anteriorly and downward, and the large terminal forces are directed upward in Case 4 (top row) and downward in Cases 3 and 5 (middle and bottom rows).**

Unlike normal loops and especially those of left atrial hypertrophy, the component parts of the P loop in right atrial hypertrophy tended to form in one plane (Fig. 9). The first part of the loop formed the maximum vector, whereas in the left atrial hypertrophy group the last part formed the maximal vector.

The latter part of the P loop was directed to the left and inferiorly, similar to normal, and lay in an extension of the plane of the first part of the loop. (3) *Direction.* The spatial orientation of the P loop of right atrial hypertrophy differed from that of the P loop of left atrial hypertrophy. The former was directed anteriorly while the latter was directed posteriorly. The direction of the maximal vector was vertical, inferior, and anterior about 30 degrees to the left of midline. In Case 4 the maximal vector was directed 15 degrees to the right of midline. The angle of the maximal vector of the P loop ranged from plus 18 degrees to plus 100 degrees (average 74 degrees) in the frontal plane; between 24 to 115 degrees (average 70.1 degrees) in the horizontal plane, and between plus 3 and plus 49 degrees (average 28.1 degrees) in the sagittal plane. The direction of the inscription of the P loop was that of the normal P loop except in one instance, i.e., counterclockwise in the frontal and horizontal planes and clockwise in the sagittal plane. The exception was Case 6 where the inscription was clockwise in the frontal and horizontal planes and counterclockwise in the sagittal plane (Fig. 15). This patient developed right atrial hypertrophy and dilatation secondary to an aorta-coronary sinus anastomosis.[9] As mentioned previously, cardiac catheterization demonstrated a shunt flow of 2 liters per minute.

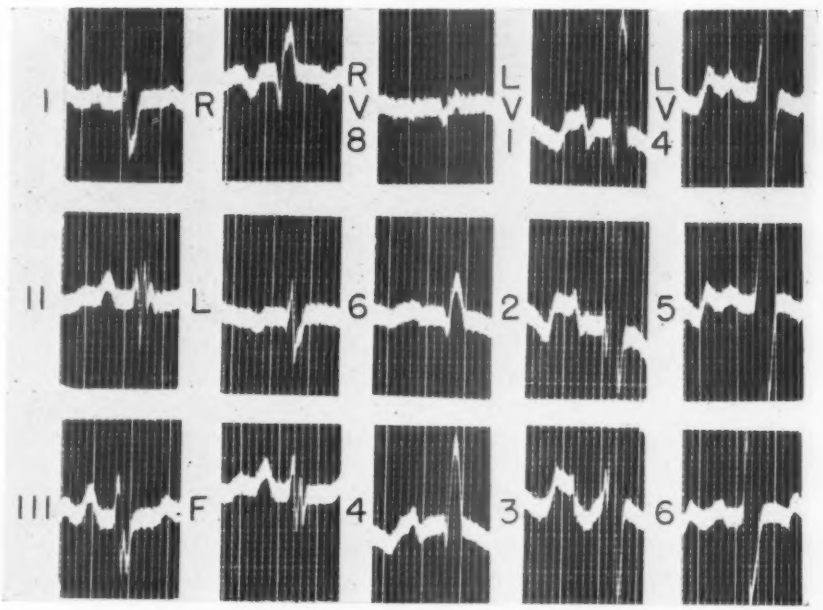


Fig. 21.—Electrocardiogram of patient with atrial septal defect and right heart failure. P vector loops are shown in middle row of Fig. 20.

*Combined Atrial Hypertrophy.*—(Figs. 20, 21, 22.) As mentioned previously, it is probable that 7 cases of the left atrial hypertrophy groups (I, Cases 2, 7; II, Cases 1, 2, 3, 6, 12) represented combined hypertrophy. They showed vectorcardiographic characteristics of both right and left atrial hypertrophy, i.e., large, early vector forces directed anteriorly, and large terminal forces directed

leftward and posteriorly. An additional five cases of combined atrial hypertrophy are mentioned here. The first case was that of a 5-year-old girl with congenital heart disease, who showed an extremely large P loop which retained some characteristics of left atrial hypertrophy. The loop however, was oriented less posteriorly than that of relatively pure left atrial hypertrophy. The second case, that of a 29-year-old woman with mitral and aortic stenosis, was similar. The third case was that of a patient who had two maximum vectors in the horizontal plane, the first directed anteriorly and the second posteriorly (Fig. 20, middle row). The patient had an interatrial septal defect with reversed flow, biatrial failure, and biatrial hypertrophy (the latter demonstrated at operation). The fourth and fifth cases each had mitral stenosis and right heart failure. The initial vector forces were directed anteriorly and were equal to the large terminal forces directed posteriorly and downward.

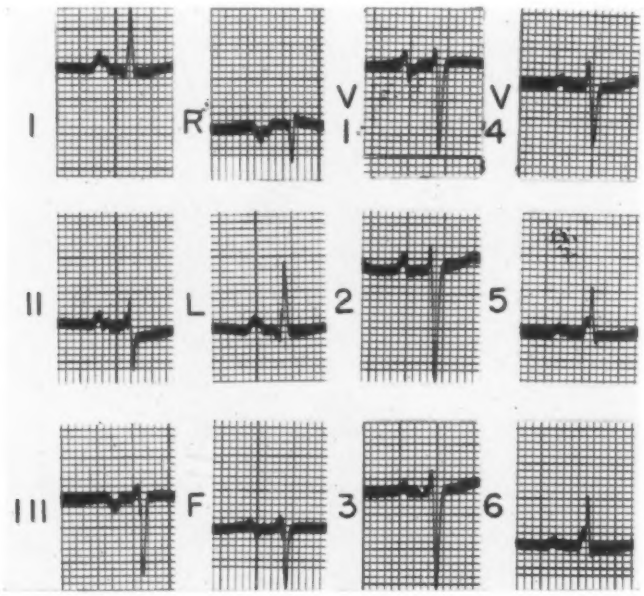


Fig. 22.—Electrocardiogram of patient with mitral stenosis and combined atrial hypertrophy. P vector loops are shown in bottom row of Fig. 20.

*P Loops of Ectopic Origin.*—(Fig. 8.) Cases 1 and 2 had either upper A-V nodal rhythm or low right atrial rhythm. The direction of inscription was counterclockwise in all three planes. When the rhythm was sino-atrial, Case 2 showed normal P loops. Cases 3 and 4 were 8-year-old children who had recurrent atrial tachycardia originating from the upper left atrium, as determined by intracardiac, unipolar, and circumferential chest leads. The loops were similar, being directed to the right, and having a clockwise inscription in the frontal and horizontal planes and counterclockwise in the sagittal plane.

*Borderline Atrial Hypertrophy.*—The vectorcardiograms of the patients with electrocardiographic evidence of probable right atrial hypertrophy, and of 3 patients with borderline left atrial hypertrophy were consistent with right and left atrial hypertrophy, respectively.

## DISCUSSION

The contour and the spatial orientation of the P loop in the normal heart, and in right and in left atrial hypertrophy is readily explained by considering the order of depolarization of the atria and the anatomical location of the predominant atrium. The superior portion of the head of the S-A node[11] is situated at the anterior margin of the junction of the superior vena cava and right auricle. The wave of excitation spreads radially at a fairly constant rate, although a recent study on the dog showed a higher rate in the taenia terminalis.[12] The onset of electrical activation of the right atrium definitely precedes that of the left atrium and appendage. This has been confirmed by studies using direct leads from the human atria.[8b] Since the left atrium is activated later, and since it is located to the left and posteriorly, the loop moves counterclockwise in the frontal and horizontal planes and, therefore, clockwise in the sagittal plane. Since the right atrium is situated more anteriorly than the left atrium, the earlier activation of the right atrium results in the first part of the atrial loop being directed anteriorly and downward; the latter portion of the P loop is directed leftward and downward, representing the activation of the left atrium.

The principal direction of rotation of the normal loop prevailed in normal patients and in all patients with right and left hypertrophy, with one exception (Case 6, RAH). This patient had iatrogenic right atrial hypertrophy, i.e., secondary to aorta-coronary sinus anastomosis. The direction of rotation of this case, clockwise in the frontal and horizontal planes and counterclockwise in the sagittal plane, was similar to the one reported elsewhere.[4] The divergence of this case is difficult to explain. Delayed right intra-atrial block, mixed electrodes, or unusual change in position (leftward and anteriorly) of a dilated right atrium could account for these changes.

Our observations that the major portion of the P loop of left atrial hypertrophy is located posteriorly and that of right atrial hypertrophy is located anteriorly are explained by the anatomical location of the hypertrophied atrium. Thus, when the right atrium which is situated right and anteriorly becomes hypertrophied, the electrical forces produced by the greater mass of atrial myocardium are directed anteriorly and downward. They overwhelm those forces produced by and pointing to the posteriorly located left atrium, pulling, so to speak, the normal loop anteriorly. Since it is likely that it takes longer for the hypertrophied chamber to be activated because of the increased mass, these anterior forces are exerted during a portion of the period of activation of the left atrium. Thus, the average duration of the P wave in right atrial hypertrophy is usually not prolonged, although the voltage is increased. The opposite situation occurs in left atrial hypertrophy. By the use of esophageal and right intra-atrial leads, the difference in right and left atrial activation has been shown to be exaggerated in left atrial hypertrophy.[13] Since the left atrium is normally activated last, the delayed activation produces a prolongation of the P wave, whereas in right atrial hypertrophy the prolongation of right atrial activation is concealed, occurring while the left atrium is activated. Thus the pulmonale P wave usually is not prolonged. The enlarged left atrium in mitral stenosis is located posteriorly and usually inferiorly. The terminal part of the loop, representing the left atrial activation, pro-

duces a bifid configuration in the frontal plane and the mitrale P wave in $aV_F$, and in Leads II and III. When the enlarged left atrium points posteriorly and superiorly, the terminal portion of the P loop is similarly directed, producing a plus-minus P wave in $aV_F$, and M-shaped P waves in Lead I and in $aV_L$. The plane of the terminal part of the P loop in left atrial hypertrophy is often twisted sufficiently to produce a figure-of-eight, most obvious in the horizontal loops. In left atrial hypertrophy the plane of the terminal part of the P loop shifts from a position almost parallel to the frontal plane of the body to one nearly perpendicular to it. In right atrial hypertrophy the enlarged initial portions of the loop also are almost perpendicular to the frontal plane. In right and in left atrial hypertrophy the enlarged atrium shifts anatomically and electrically toward the midline, in the former anteriorly, and in the latter posteriorly.

The two subjects with upper left atrial pacemakers showed similar electrocardiograms and vectorcardiograms. The P waves were negative in $aV_L$, in Lead I, and in left chest leads; positive in right chest leads and in $aV_F$; and slightly negative or diphasic (plus-minus) in $aV_R$. The vectorcardiogram showed clockwise rotation in the frontal and horizontal planes and counterclockwise in the sagittal plane. Thus, rotation was opposite to that of the normal and was readily explained by the earlier activation of the left atrium.

In the two cases with nodal or lower atrial rhythm, the impulse started in the region of the A-V node. The direction of inscription was not clockwise in the frontal and horizontal planes, as would be expected, but was counterclockwise. This was surprising, since one would reasonably expect the right atrium to be activated first to produce clockwise rotation. The possibility existed that the focus was in the lower left atrium near the lower part of the interatrial septum.

In combined hypertrophy, as in mitral stenosis with elevation of pressure in the left atrium and the right heart, or as in interatrial septal defect with right heart failure and right-to-left flow, the electrical forces produced by both atria were increased. Thus, the magnitude, as well as the duration, of the anterior (right) and posterior (left) forces was increased.

In hypertrophy the Ta displacement and its equivalent (opening of the P vector loop) increased in magnitude and changed location. In normal patients the loop was slightly open, was located in the region of minus 90 degrees in the frontal and sagittal planes, and thus was closed in the horizontal plane. The failure of the P loop to return to the isoelectric point is not apparent in other methods of recording and lesser amplification. In right atrial hypertrophy the Ta shifted posteriorly and to the right about 25 degrees, and lay about 180 degrees from the maximum vector which pointed anteriorly and leftward (Fig. 2). A similar relationship existed in left atrial hypertrophy. Ta shifted anteriorly and to the right, while the maximal P vector shifted posteriorly and leftward. Thus, Ta, which represented repolarization of the atria, had a similar orientation to the wave of atrial depolarization as the ST-T complex had to ventricular activation, i.e., approximately 180 degrees from the maximal QRS vector.

An opinion has been expressed that "the pattern of pulmonale P (right atrial hypertrophy) is not caused by unilateral auricular hypertrophy but may be primarily the result of abnormal cardiac rotation or of auricular dilatation

which allows the electrical effects of both auricles to be deflected backward and toward the left leg."[13] Our observations did not support the above view. Considering the electrode position of the cube reference system, and considering that all planes showed large P loops in right atrial hypertrophy, the pulmonale P did not seem to be produced merely by the rotation of the heart, but rather was due to an increase of the electrical activity of the right atrium. Acute right atrial dilatation may produce pulmonale P waves, representing strain rather than hypertrophy. Whether the increased electrical activity is produced by hypertrophy or strain is immaterial, since the distinctive loop of the pulmonale P is produced by increased electrical activity of the right atrium.

Our criteria of pulmonale P waves in the electrocardiogram may not receive universal acceptance (narrow peaked P waves in Leads II, III, and $aV_F$, and upright, large peaked P wave in $V_1$). Other authors[8,10,14,15] have stated that the P wave in $V_1$ is diphasic, negative, or rarely altered. In our personal experiences, especially in a tuberculosis hospital, we found positive P waves in $V_1$ and $V_2$ in the fourth or fifth intercostal spaces to be characteristic of cor pulmonale, proved at autopsy. We do not deny that some cases of cor pulmonale show diphasic (plus-minus) forms in $V_1$ and sometimes even a negative wave. In such cases, differentiation from the second type of left atrial hypertrophy becomes difficult.

The contour of the P wave depends greatly upon the position of the exploring chest electrodes in relation to the atria: thus, if $V_1$ and $V_2$ are at the level of the S-A node, P will be zero or negative; if over the mid-right atrium diphasic; if at the level of the A-V sulcus, positive. Because of the distant position of the vectorcardiographic electrodes, the P vector loops present more constant features. The importance of the position of the exploring electrocardiographic electrode was well shown in Case 1 of the right atrial hypertrophy group (Figs. 18, 19). This patient had autopsy-proved chronic cor pulmonale. The P vector loop was typical of right atrial hypertrophy. The P waves were diphasic in $V_1$ and negative in the other chest leads. It is difficult to explain the configuration unless one assumes that the electrode positions were significantly above the site of impulse formation, i.e., above the S-A node, or that a transient change in cardiac mechanism had occurred. The electrocardiograms taken a few days previously, and those later, showed typically pulmonale P waves (Fig. 19). The placement of the electrodes in the vectorcardiographic reference system obviates the above difficulties.

*The Value of the Atrial Vectorcardiogram.*—The technique of electrical dissection of the P wave and the constancy of the normal patterns facilitates the early detection of the abnormal and, especially, the distinction between right and left atrial hypertrophy. This is true especially in patients with Type II left atrial hypertrophy.

The superiority of the "dissected" vectorcardiogram lies in the ease with which deviations from the normal pattern can be detected. The same information admittedly can be obtained by the synthesis of multiple, simultaneous chest and limb leads. For example, the P waves of the electrocardiogram of Case 4 of the "practically normal" group showed no abnormality, although the P vector loop showed a posterior location beyond the normal range. A record taken 11 days

previously during an acute myocardial infarct showed P vector loops of left atrial hypertrophy, and the electrocardiograms showed Type II left atrial hypertrophy. Thus, the vectorcardiogram retained some features of left atrial hypertrophy or "strain" not obvious in standard limb leads and chest leads. The features of left atrial hypertrophy were distinctive in the vectorcardiograms of the patients with borderline left atrial hypertrophy, but not clearly shown in the electrocardiograms. Two of the patients had made a striking improvement after mitral commissurotomy, and the third one a recovery from a large myocardial infarct with left heart failure. The vectorcardiogram during recovery showed a decrease of posterior direction in this group, consistent with some residual left atrial hypertrophy. Wenger[16] also observed a considerable tendency of the P vector loop to move anteriorly after commissurotomy.

Of course, the electrocardiographic diagnosis of such conditions depends upon the strictness of adherence to numerical criteria. If the importance of contour is recognized, many of the above diagnoses, conspicuous in the vectorcardiogram, may also be made from the electrocardiogram. Wenger[16] concurs in the view that the atrial vectorcardiogram gives a clearer picture of the activation of the atria than does the study of the electrocardiogram alone.

## SUMMARY

1. The isolated P vector loops were obtained by the technique of electrical dissection (differential vectorcardiography), in 81 cases: 16 normal cases; 15 cases with practically normal P waves; 23 cases with left atrial hypertrophy; 3 cases with borderline left atrial hypertrophy; 1 case with probable right atrial hypertrophy; 14 cases with right atrial hypertrophy; 5 cases with combined atrial hypertrophy; and 4 cases with ectopic origin of the P wave.

2. Exclusive of P vector loops of ectopic origin, the direction of inscription of the P vector loop, in both healthy and diseased patients was principally counterclockwise in the frontal and horizontal planes, and clockwise in the sagittal plane.

3. The normal P loop was small, closed, or slightly open, and directed almost vertically inferiorly. The P loop of left atrial hypertrophy was larger and more opened, and the maximal vector was directed posteriorly, inferiorly and leftward. The P loop of right atrial hypertrophy was the largest and most widely opened and its maximum vector was directed anteriorly and inferiorly.

4. The Ta vector was located about 180 degrees from the maximal P vector in hypertrophy. The physiologic significance of the morphology of the P vector loop was discussed.

5. The P vector loop is often helpful clinically in the differentiation between right and left atrial hypertrophy, when standard electrocardiograms are equivocal.

6. The superiority of the isolated P vector loops lies in the ease with which deviations from the normal pattern can be detected.

We are indebted to Miss Marlyn Guffey, Miss Rita McGreevy, and Mr. Robert Hamlin for assistance in the preparation of the illustrations and the manuscript.

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# THE TWO MAIN QRS VECTORS IN THE FRONTAL PLANE IN ELECTROCARDIOGRAMS OF COMPLETE LEFT BUNDLE BRANCH BLOCK

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IN ELECTROCARDIOGRAMS of cases with complete left bundle branch block (LBBB), two main QRS vectors may be distinguished in the frontal plane. The first of these can be taken to indicate the main direction of activation of the right ventricle, the second one that of delayed left ventricular activation. The object of the present study was to investigate the incidence in the various directions of these two vectors and the angles formed by them.

## METHOD

From about 5,000 records, 50 tracings of uncomplicated LBBB were selected according to the criteria established by Wilson and associates.[1] All were 12-lead records (I, II, III, $aV_R$, $aV_L$, $aV_F$, and $V_1$ through $V_6$) taken with a direct-writing electrocardiograph.* Clinical data or post-mortem findings were not considered.

The direction of the two main vectors was graphically represented in a circular diagram, based on Einthoven's triangle.[2] It consists (Fig. 1) of six concentric circles representing, from within outwards, Leads I, II, III, $aV_R$, $aV_L$ and $aV_F$. Each circle is composed of a "positive" semicircle (white) and a "negative" semicircle (gray). Vectors situated in the "positive" semicircle yield a positive deflection in the respective lead; those in the "negative" semicircle a negative one; and those coinciding with the dividing line between these semicircles zero (transitional) deflections. If no zero potential is recorded in any lead, the direction is, for the sake of simplicity and uniformity, assumed as being midway at 30 degrees in the appropriate sector. The circle is thus subdivided into 24 sectors of 15 degrees each, as indicated at the periphery.

The tracing reproduced below the diagram was selected at random by way of example. The black arrow head in the diagram indicates the first main QRS vector in the frontal plane, and the white arrow head indicates the second one; the angle formed by these vectors is seen to be 90 degrees.

## RESULTS

The directions of these two vectors and the angles formed by them are given in Table I. QRS′ indicates the first, and QRS″ the second main QRS vector.

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The incidence in per cent of these two vectors in the various directions, and the incidence in per cent of the various angles are diagrammatically illustrated in Figs. 2 and 3. The curve of the first main vector occupied mainly the left lower quadrant; the second the left upper quadrant. Most angles (94 per cent) did not exceed 90°, the average being 45°.

TABLE I. DATA AS TO QRS′ AND QRS″ VECTORS AND THEIR DEFINED ANGLES IN 50 LBBB ELECTROCARDIOGRAMS

| RECORDS | QRS′ (DEGREES) | QRS″ (DEGREES) | ANGLE (DEGREES) | RECORDS | QRS′ (DEGREES) | QRS″ (DEGREES) | ANGLE (DEGREES) |
|---|---|---|---|---|---|---|---|
| 1 | −15 | −30 | 15 | 26 | +30 | −60 | 90 |
| 2 | +45 | +45 | 0 | 27 | 0 | −15 | 15 |
| 3 | +30 | −45 | 75 | 28 | +15 | −45 | 60 |
| 4 | +45 | +15 | 30 | 29 | +75 | −75 | 150 |
| 5 | −15 | −45 | 30 | 30 | +15 | −45 | 60 |
| 6 | +15 | −45 | 60 | 31 | −15 | −45 | 30 |
| 7 | +45 | −45 | 90 | 32 | −45 | −45 | 0 |
| 8 | +45 | +15 | 30 | 33 | +60 | −45 | 105 |
| 9 | +45 | −45 | 90 | 34 | −15 | −45 | 30 |
| 10 | +15 | −45 | 60 | 35 | 0 | −45 | 45 |
| 11 | +45 | −15 | 60 | 36 | −60 | −60 | 0 |
| 12 | +30 | +15 | 15 | 37 | +15 | −15 | 30 |
| 13 | 0 | −45 | 45 | 38 | +45 | −45 | 90 |
| 14 | 0 | −15 | 15 | 39 | +45 | −45 | 90 |
| 15 | +45 | −45 | 90 | 40 | +45 | −15 | 60 |
| 16 | +75 | −75 | 150 | 41 | 0 | −45 | 45 |
| 17 | 0 | −75 | 75 | 42 | −15 | −45 | 30 |
| 18 | +45 | +45 | 0 | 43 | +45 | 0 | 45 |
| 19 | +15 | −15 | 30 | 44 | 0 | −45 | 45 |
| 20 | +30 | +30 | 0 | 45 | +45 | −15 | 60 |
| 21 | −15 | −15 | 0 | 46 | −15 | −45 | 30 |
| 22 | −15 | −45 | 30 | 47 | +45 | −15 | 60 |
| 23 | −45 | −75 | 30 | 48 | +45 | +45 | 0 |
| 24 | +30 | 0 | 30 | 49 | −30 | −45 | 15 |
| 25 | 0 | −45 | 45 | 50 | −15 | −15 | 0 |

## DISCUSSION

The curve of the first main QRS vector occupied nearly the same place as those of the first main QRS vector from complete right bundle branch block, RBBB,[2] and those of normal ÂQRS.[3] In vectorcardiography the above evidence would indicate that the initial portion of the QRS form in the frontal plane from bundle branch block, either left or right, occupied nearly the same place as that from normal records. Burch and associates[4] observed that in pure RBBB the form and orientation of the initial QRS portion in space was indistinguishable from that of normal records.

The curve of the second main QRS vector from LBBB is located extremely far to the left. It is very distant from the curve of the first main QRS vector from RBBB[2] and left of the ÂQRS curve from left ventricular hypertrophy.[3] The route of activation of the blocked left ventricle is abnormally altered toward the left.

It should be noted that the curve of the second main QRS vector from RBBB is located extremely far to the right. It is very distant from the curve of the first main QRS vector from LBBB, and to the right of the ÂQRS curve from right ventricular hypertrophy.[3] The route of activation of the blocked right ventricle is abnormally altered toward the right.

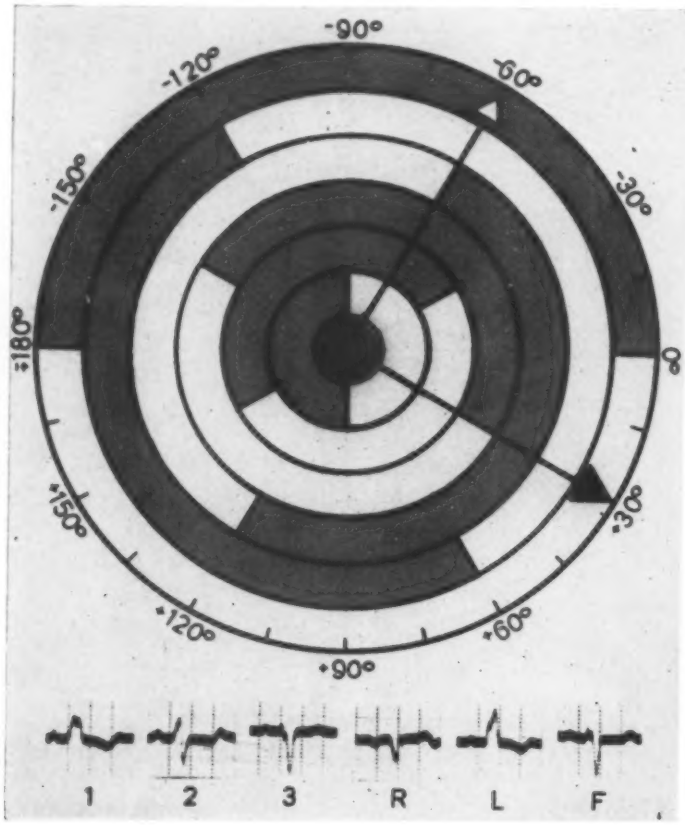


Fig. 1.—A circular device for the RLF plane.

## SUMMARY

1. The incidence in the various directions of the two main QRS vectors in the frontal plane in LBBB was investigated. The first main QRS vectors had nearly the same directions as the normal ÂQRS, the second one occupied the left upper quadrant. The angles formed by the two vectors usually did not exceed 90°, and was 45° on the average.

2. The incidence of LBBB was about 1 per cent; its incidence to RBBB was as 1 to 2.

3. The general direction of activation of a single normal ventricle, either left or right, was nearly the same as normal ÂQRS. The route of activation of a blocked ventricle, either left or right, was abnormally altered; toward left in LBBB, toward right in RBBB.

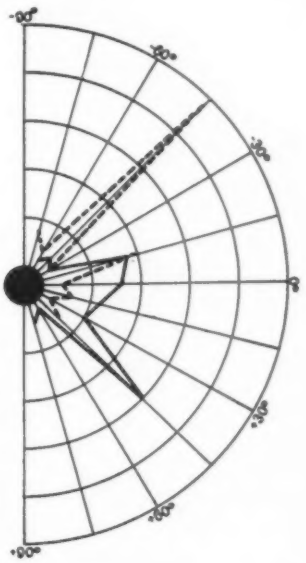

Fig. 2.—Diagram showing the incidence in per cent in the various directions of the first (solid line) and second (broken line) main QRS vectors, the 5 concentric semicircles indicating, from within outwards, 10, 20, 30, 40, and 50 per cent, respectively.

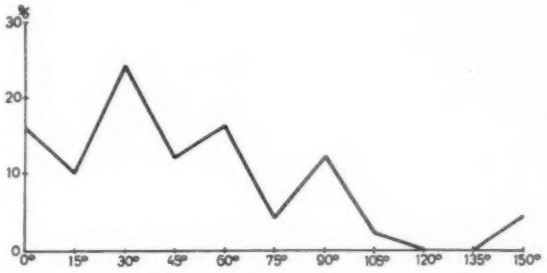


Fig. 3.—Diagram showing the incidence of angles formed by the two main vectors. Abscissa: angles; ordinate: per cent.

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# INTERACTIONS BETWEEN ATRIA AND VENTRICLES DURING INTERFERENCE-DISSOCIATION AND COMPLETE A-V BLOCK

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THE terms "A-V dissociation" and "complete A-V block" seem to imply that divorce between the cardiac partners is absolute. But, as van Buchem[1] commented in 1929, "Atria and ventricles frequently do not function so independently in complete A-V block as one might be inclined to think." The same may certainly also be said of interference-dissociation. One of the purposes of this communication is to emphasize that independence between atria and ventricles during complete A-V block or interference-dissociation is often far from absolute.

A second purpose is to draw attention to the unfortunate circumstance that high authorities differ in their use of the two terms, "dissociation" and "interference."

*Dissociation* is used by some to embrace any rhythm in which atria and ventricles beat independently, and thus includes both interference-dissociation and complete A-V block. Others, and these are probably the majority, use the term as synonymous with interference-dissociation and by its use exclude complete A-V block. In this review the term A-V dissociation will be used to signify independent action of atria and ventricles regardless of the mechanism producing that independence.

*Interference* is given two distinct and opposed interpretations. One school reserves it for the electrophysical phenomenon which may be defined as "the mutual extinction of two excitation waves that meet in any portion of the heart."[2] This use implies that the dissociation results from the phenomenon of interference, as defined above, between S-A and A-V impulses. The second school uses the term in a simpler sense to mean an interference by occasionally conducted S-A impulses with the otherwise independent rhythm of the ventricles; and such conducted beats are not uncommonly referred to by members of this school as "interference beats."

This discrepancy in the use of interference is particularly unfortunate because the two interpretations seriously conflict. For the interference beats of the second school are the only beats in which the phenomenon of interference, as interpreted by the first school, plays no part; rather they are isolated normal beats which punctuate a series of abnormally dissociated cycles. Without necessarily implying that it is more correct, but because one or other usage must be consistently adopted for the purpose of this discussion, that of the first school will be here employed.

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The several ways in which atria and ventricles can influence one another, despite the presence of A-V dissociation, may be listed as follows: (A) during interference-dissociation: (1) ventricular capture, (2) synchronization; (B) during complete A-V block: (1) retrograde beats (atrial capture), (2) fortuitous forward conduction, (3) ventriculoatrial arrhythmia, (4) synchronization.

Each of these phenomena will be described and illustrated. Where authors have applied differing terms to the same phenomenon, the synonymous terms will be presented side by side in an attempt to clarify the terminology in this rather complex field.

## VENTRICULAR CAPTURE DURING INTERFERENCE-DISSOCIATION

During A-V dissociation due to interference, whenever the atrial impulse arrives at the junctional tissues after the end of their refractory period, the impulse is conducted and ventricular capture[3] takes place. In terms of the electrocardiogram this occurs whenever the P wave falls far enough beyond the QRS complex for the atrial impulse to be conducted. When occasional beats are thus conducted, the mechanism has been called incomplete A-V dissociation to distinguish it from complete A-V dissociation where no conducted beats occur.[3] In general, for ventricular capture to take place, certain conditions must be fulfilled[4,5]: (1) atria must be beating slower than ventricles (so that the P wave can "overtake" and pass the QRS complex); (2) the capacity for forward A-V conduction must remain unimpaired; (3) retrograde V-A block must be present (protective or entrance block) so that the slower-beating atria are protected from control by the faster beating ventricles.

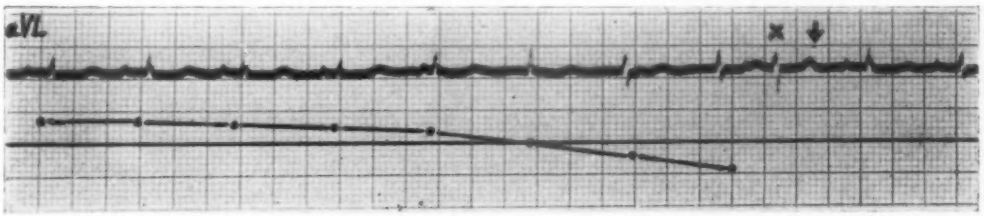


**Fig. 1.—Interference-dissociation with ventricular capture (*x*). The capture beat shows ventricular aberration. As a visual aid to the changing A-V relationships, and for comparison with Fig. 5, the P-R and R-P intervals are plotted in the graph below the tracing; P-R intervals are plotted as distances above the horizontal line, while R-P intervals are plotted below it.**

Examples of the classical form of interference-dissociation with ventricular capture are shown in Fig. 1 and in Lead $V_4$ of Fig. 2. The conducted impulse disrupts the basic ventricular rhythm in two ways: (1) by inducing a ventricular response that is premature; (2) by extinguishing in its passage through the conducting tissues the A-V impulse that was beginning to form and which, if it had not been for the disturbing S-A impulse, would have initiated a ventricular response after the usual R-R interval, i.e., at the arrow in Fig. 1.

Rarely, the regular ventricular rhythm is differently interrupted. Instead of a ventricular beat appearing prematurely, the ventricles fail to be captured, yet the next QRS complex is displaced to where it would have appeared following a "capture" beat. The result is an unexpectedly long R-R interval (Lead II

in Fig. 2). This is explained by postulating block in the junctional tissues below the site of the ventricular pacemaker. The S-A impulse penetrates and discharges the nodal pacemaker but cannot proceed to activate the ventricles because of blockade lower down. The forming nodal impulse, however, has been extinguished and the process of impulse formation has to begin anew. Following Langendorf,[6] Katz and Pick[3] refer to this phenomenon as one form of concealed conduction, while Gentile[7] has employed the terms dissociation with double interference and dissociation with ladder-type interference.

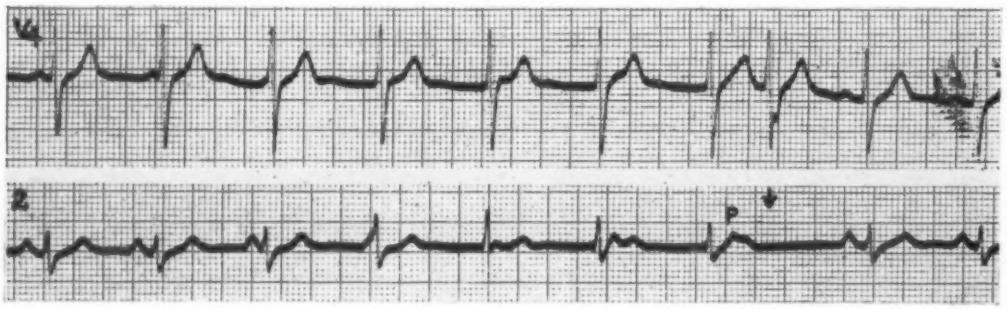


Fig. 2.—Lead $V_4$ shows interference-dissociation with ventricular capture. Lead II shows almost identical A-V relationships except that the capture beat expected (at the arrow) failed to appear. For fuller description see text.

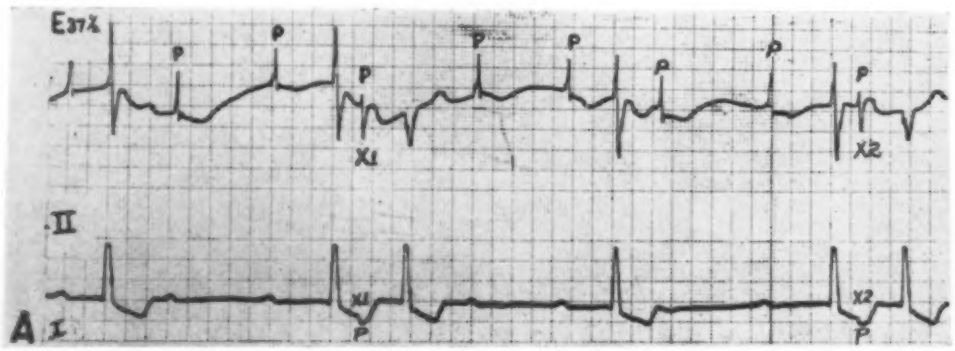


Fig. 3.—Complete A-V block. An esophageal tracing is shown in the upper strip with simultaneous Lead II below. Following the second ventricular complex in each lead, an early retrograde P wave appears, presumably due to retrograde conduction of the ventricular impulse into the atria. This retrograde P wave is in turn followed by a ventricular response (which shows aberration in the esophageal lead). This is presumably a conducted beat, A-V conduction having occurred because the impulse fortuitously arrived at the junctional tissues during their phase of supernormal excitability which was enough to overcome the otherwise effective A-V block. This unusual sequence represents a form of reciprocal rhythm. The penultimate ventricular complex in each lead is followed by an identical sequence. (From Bellet: Clinical Disorders of the Heart Beat, Lea & Febiger, 1953, with permission of author and publishers.)

## RETROGRADE CONDUCTION DURING COMPLETE A-V BLOCK

On occasion, in the presence of complete A-V block, the ventricular impulse is propagated backward into the atria so that a retrograde P wave is inscribed (Fig. 3). This represents, in terms of mechanism, the exact opposite of interference-dissociation with ventricular capture.[4,8] Here the slower ventricles dis-

rupt the basic rhythm of the faster beating atria by inducing them to beat prematurely, and this may be analogously called atrial capture.[3]

### FORTUITOUS FORWARD CONDUCTION IN COMPLETE A-V BLOCK

By definition, complete A-V block precludes the possibility of forward A-V conduction. However, in the presence of virtually complete A-V block, the passage of an atrial impulse which fortuitously arrives at the junctional tissues during their phase of supernormal excitability[9] may be thereby facilitated and therefore able to penetrate to and activate the ventricles. Such an atrial impulse may theoretically arise as (a) the regular sinus impulse, (b) an ectopic atrial impulse, or (c) a retrograde beat from the ventricles (Fig. 3). When such a retrograde beat retraces its steps and again invades the ventricles as in Fig. 3, the resulting V-A-V sequence may be regarded as a form of reciprocal rhythm.

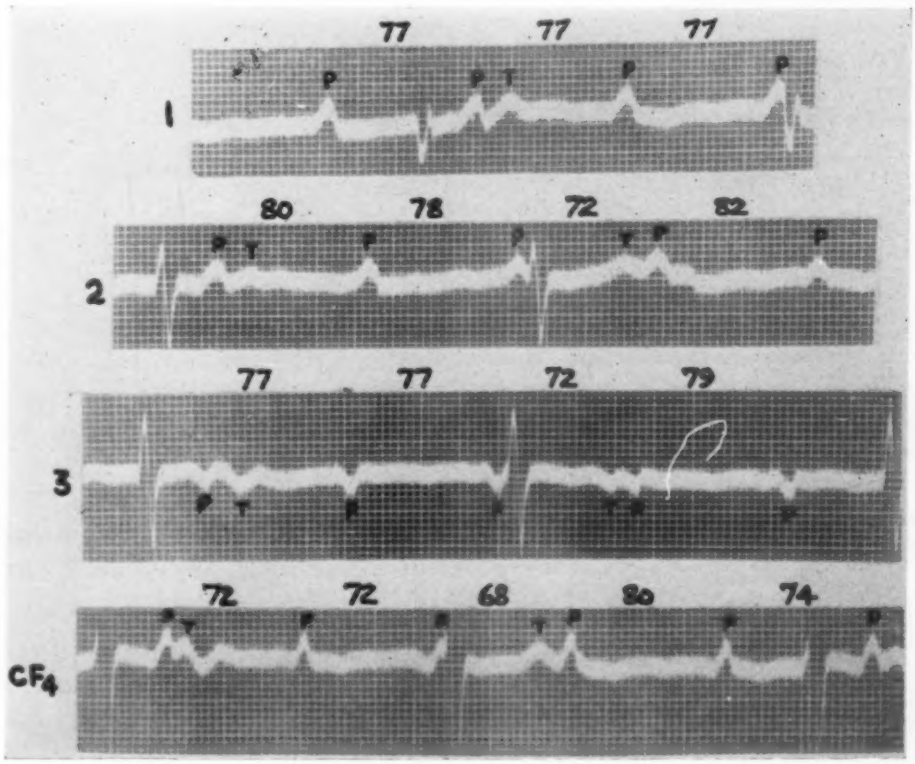


**Fig. 4.—Complete A-V block with V-A arrhythmia. P-P intervals are recorded in hundredths of a second above each strip. Note that in Leads II, III, and $CF_4$, the P-P intervals containing QRS complexes are notably shorter than those not containing QRS complexes.**

### VENTRICULOATRIAL ARRHYTHMIA DURING COMPLETE A-V BLOCK

In this phenomenon, to which attention was first drawn by Erlanger and Blackman[10] in 1910, the P-P intervals containing QRS complexes (P-R-P intervals) are measurably shorter than other P-P intervals. This is evident in Leads II, III, and $CF_4$ in Fig. 4. This disturbance of atrial rhythm has also been called sinus allorhythmia[11] and ventriculophasic sinus arrhythmia.[3]

Of 147 cases of complete A-V block collected from the literature and reviewed by Rosenbaum and Lepeschkin,[12] this phenomenon was present in 42 per cent. On the other hand, P-R-P intervals paradoxically longer than P-P intervals were present in 3.5 per cent. In 54.5 per cent no significant difference between P-R-P and P-P intervals was observed.

The cause of such dislocation of atrial rhythm, when it occurs, is not positively known and has been disputed for decades. Rosenbaum and Lepeschkin[12] refer to the shortening of the P-R-P interval as the positive chronotropic effect of ventricular contraction, and they attribute it to the influence of mechanical traction on the atria leading to stimulation of the S-A node and consequent earlier impulse formation. Lengthening of the subsequent P-P interval is referred to as the negative chronotropic effect; this they believe to be a vagal effect initiated by the arterial pulse wave which, through pressoreceptors, produces reflex delay in impulse formation by the S-A node.

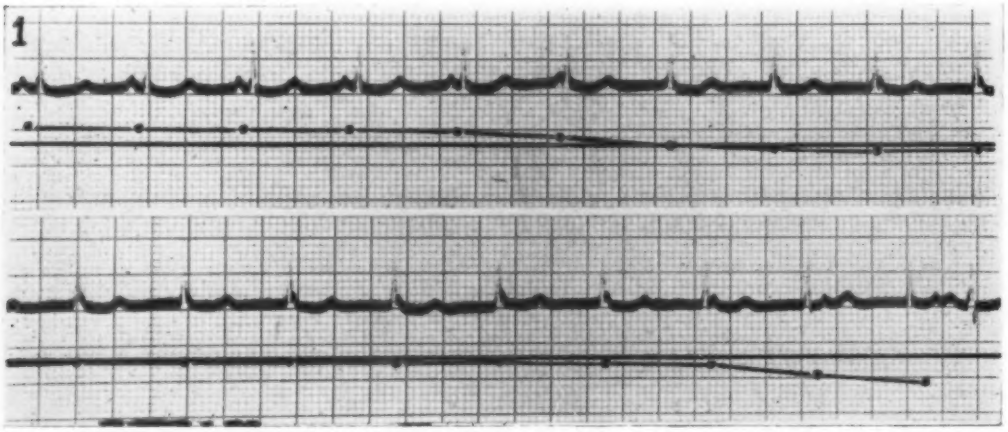


**Fig. 5.—Interference-dissociation with synchronization. As in Fig. 1, in the graph below the tracing P-R intervals are plotted above the line while R-P intervals are plotted below it. When P waves and QRS complexes approach each other they remain linked together—the P wave appears to experience "difficulty in getting past" the QRS complex. Compare with Fig. 1.**

## SYNCHRONIZATION DURING A-V DISSOCIATION AND COMPLETE A-V BLOCK

*Synchronization* is a phenomenon that has long been recognized to occur in juxtaposed tissues possessing inherent rhythmicity. In brief, if rhythmigenic tissues are placed in contact, provided that their intrinsic rhythms are not greatly dissimilar, they will often settle down to a common rate of discharge and pulsate together. This capacity was demonstrated in nervous tissues many years ago but has only recently been found to apply to cardiac muscle. Segers[13] showed that synchronization developed between fragments of frog's heart and also between atria and ventricles in experimental complete A-V block in the frogs' heart. He later reported an example of synchronization occurring during complete A-V block in the human heart.[14] When periods of synchronization were brief, he applied the term *accrochage*. His work has recently been reviewed in detail[15] and will therefore not be enlarged upon here.

It must be emphasized that A-V synchronization does not simply mean the synchronous, or almost synchronous, contraction of atria and ventricles.

This occurs in A-V nodal rhythm which is not a form of synchronization. In synchronization two separate and autonomous pacemakers control the heart yet form impulses simultaneously, or almost so. The phenomenon is analogous to two individuals walking. During synchronization they walk side by side, but each remains autonomous and either may change pace and so leave his companion's side. The tracings and graphs in Figs. 1 and 5, which are from the same patient, should be compared with this walking simile in mind. In Fig. 1 one walker overtakes and passes the other without pausing; in Fig. 5 one falls in step with the other for several paces before pulling ahead. Thus the record in Fig. 1 illustrates interference-dissociation without synchronization, whereas that in Fig. 5 represents the phenomenon of synchronization (or accrochage) occurring during dissociation. Some influence, electrical or mechanical or both, for a time holds the atria and ventricles beating in phase. It seems likely that this phenomenon occurs much more frequently than it has so far been recognized.[16]

If synchronization develops during complete A-V block, because of the usually prevailing rates of atria and ventricles, the ventricles are likely to synchronize with every second or third atrial beat. For an example of this, reference may be made to the case reported by Segers.[14]

## SUMMARY

1. It is emphasized that during interference-dissociation and complete A-V block atria and ventricles are not always entirely independent.

2. Ventricular capture or A-V synchronization may occur during interference-dissociation.

3. Retrograde conduction to the atria, fortuitous forward conduction, ventriculoatrial arrhythmia, or A-V synchronization may occur during complete A-V block.

4. Each of these phenomena is described and illustrated.

5. Attention is drawn to discrepancies in usage of the important terms "dissociation" and "interference."

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# PRESSURE-"VOLUME" DIAGRAMS OF THE LEFT VENTRICLE OF MAN

## (A Preliminary Report)

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AS INDICATED previously,[1] the mechanical work of the heart can be determined most accurately from direct recording of the pressure-volume (P-V) diagrams for each ventricle during the cardiac cycle. Engineers have used P-V diagrams to study the performance of pumps and engines. Valvular dysfunctions, velocity of blood flow, and other such factors will not influence the measurement of the time-course of ventricular work determined from these diagrams. If the time-course of heat production or oxygen consumption of the heart could be measured simultaneously with the P-V diagrams of the ventricles during a cardiac cycle, then the time-course of cardiac efficiency could be obtained. Unfortunately, this is not yet possible, although Rushmer[2] has attempted to record the pressure-circumference relation of the left ventricle of dogs.

It is evident from previous studies[1,3] that the configuration of the pressure-volume diagram must vary quantitatively with changes in the mechanical behavior of the respective chambers of the heart as a result of physiologic alterations in health and disease. For this reason, it seemed important to attempt to record the time-course of ventricular work in health and disease. This preliminary report describes curves obtained thus far for the left ventricle of man and indicates the problems yet to be solved.

### METHODS AND RESULTS

With the subject lying on a fluoroscopic table, the *left ventricular pressure* was recorded by means of a pressure transducer connected to a 17-gauge needle, the beveled end of which had been inserted into the ventricular chamber percutaneously from a point on the chest just to left of the apex of the heart. The amplified signal from the transducer was made to activate the vertically deflecting plates of a cathode-ray oscilloscope. The pressure trace was calibrated with a mercury manometer.

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The time-course of *left ventricular "volume"* was recorded indirectly by the roentgenographic densimetric method described by Ring and associates,[4] with the x-ray beam penetrating the left ventricle and with the subject rotated about 30 degrees in the left anterior oblique position. The multiplier phototube was positioned over the left ventricle fluoroscopically and was activated by the x-ray induced light flux from a piece of fluorescent screen fastened to its envelope. The signal from the phototube was amplified and fed to the horizontally deflecting plates of the oscilloscope. The densimetric trace was calibrated in units of "volume"* change by intermittently inserting pressedwood (masonite board, 1/8 inch thick) into the path of the x-ray beam and converting the resulting change in deflection to the relative thickness of blood, as described previously by Ring and co-workers.[4]

Four patients with aortic valvular disease were studied. In Fig. 1 is shown the pressure-"volume" diagram of one such patient; in Fig. 2 the time-course

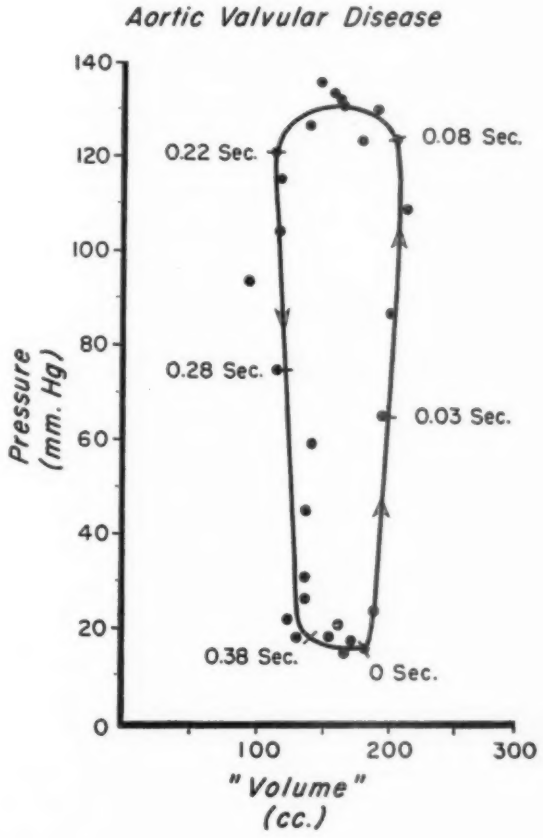


**Fig. 1.—Pressure-"volume" diagram of the left ventricle, during a single cardiac cycle, in a patient with rheumatic aortic valvular stenosis and insufficiency. The points, 1/60 second apart, are shown as they were recorded directly by the cathode-ray oscilloscope. The "volume" component is only an approximation of true volume of the left ventricle, as indicated in the text.**

*Obviously, this function is not true volume but only an approach to the quantitative value of volume, which is the factor ultimately desired.

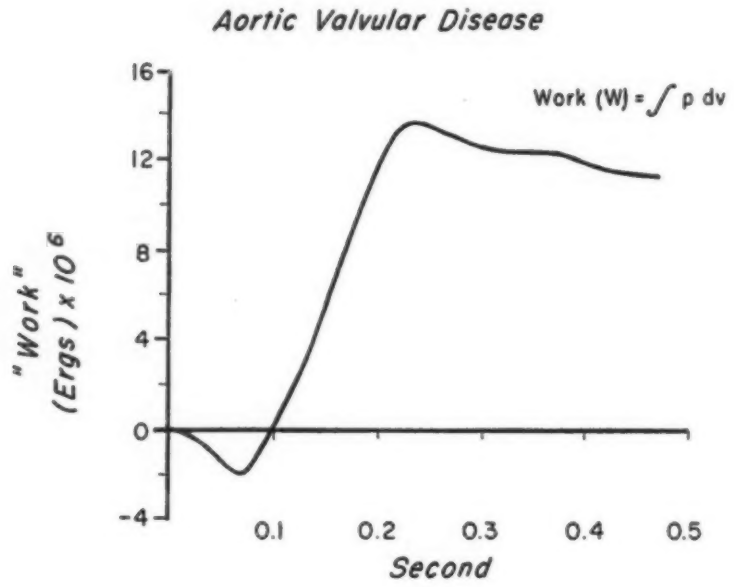


Fig. 2.—Time-course of accumulated "work" of the left ventricle derived from the pressure-"volume" diagram shown in Fig. 1.

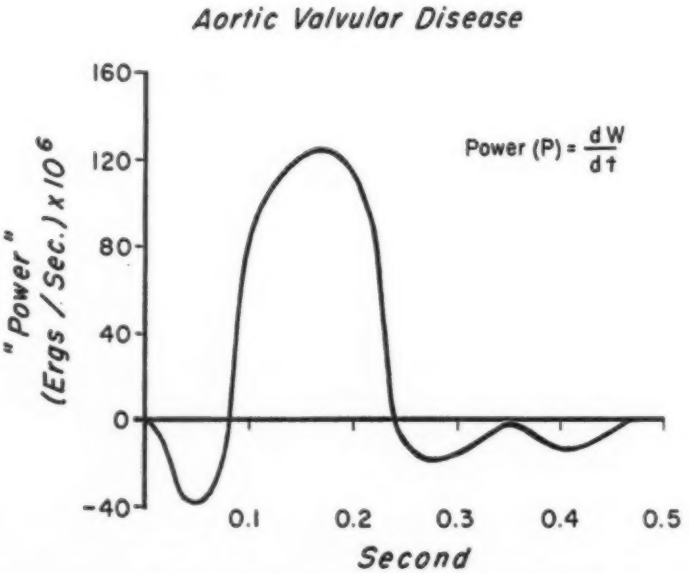


Fig. 3.—Time-course of "power" of the left ventricle derived from the "work" curve shown in Fig. 2.

of accumulated mechanical "work"; in Fig. 3 the time-course of "power"; and in Fig. 4 the time-course of the first derivative of power. The time-course of "tension" on the wall of the ventricle (Fig. 5) was calculated by a method previously described.[3]

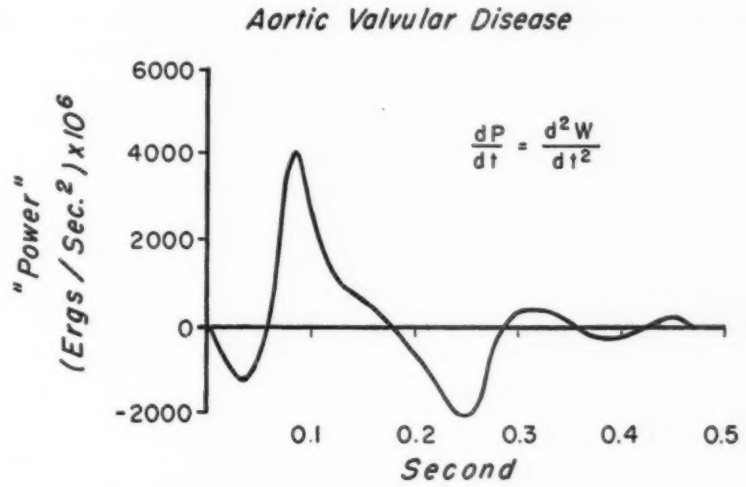


Fig. 4.—Rate of change of "power" of the left ventricle for the cardiac cycle shown in the preceding illustrations.

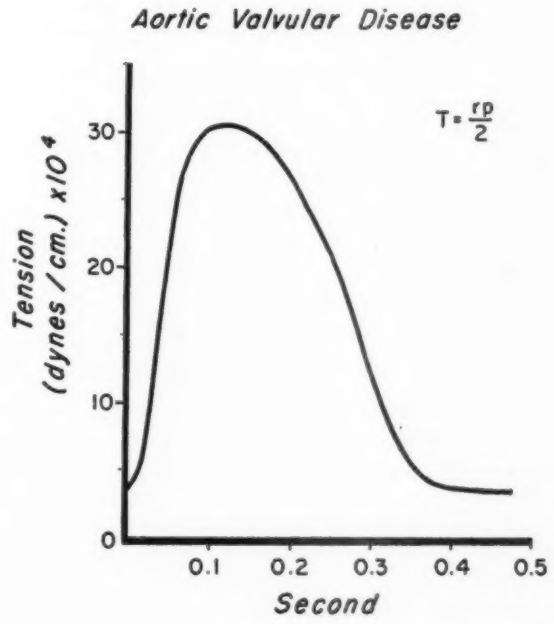


Fig. 5.—Time-course of "tension" of the left ventricle for the cardiac cycle shown in the preceding illustrations.

## COMMENT

The pressure-"volume" diagram shown in Fig. 1 is only an approximation of the true curve for the left ventricle, because deflection indicative of volume merely approximates the true volume-time for the ventricular cycle, as indicated hereinafter. The "work" and "power" curves, which were derived from the pressure-"volume" diagram, are therefore also only approximations. The configuration of the pressure-"volume" diagram is essentially what might be expected for a subject with aortic valvular stenosis and insufficiency and would be expected to be more vertical than that for the normal subject.[1,3]

The recording of pressure was sufficiently accurate to be considered satisfactory, but the necessity for left ventricular puncture in itself eliminates routine recording of pressure-"volume" diagrams by this method. Whereas right and left ventricular catheterization by means of left atrial puncture or any similar approach likewise precludes this type of study for routine purposes, they do permit accurate recording of pressure for pressure-volume diagrams of any of the chambers of the heart of man.

## SUMMARY

Pressure-"volume" diagrams of the left ventricle of intact man have been recorded. The "volume" component of the trace was obtained indirectly and, therefore, only approximates the true value. With improvements in the method, determination of this parameter should become more accurate. Developmental studies to permit simultaneous recording of the pressure-"volume" diagram and the time-course of pressure, volume, power, accumulated work, and other functions are in progress.

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# POSTURE, THE MANIFEST HEART VECTOR, AND THE VOLUME CONDUCTOR

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THIS report describes postural variation of the manifest vector as a function of postural changes in the volume conductor. Both variables are related to a common denominator, the effective axes of the recording leads. This is a practical method for handling the realities of the body as a volume conductor in the living subject.

Einthoven[1] was the first to study the effects of posture on the electrocardiogram. In Reference 1 on pages 182 and 209, he shows Lead I taken with the patient lying on the left and right sides. The S is very small in the left posture and large in the right. If we assume the patient was normal and the mean QRS axis was directed inferiorly, this is equivalent to a deviation of the manifest vector to the right when the subject lies on the right side. Of 11 of his subjects, 6 showed this behavior. To explain it, he assumed that the heart rotated about a longitudinal axis whereby the S vector turned from a sagittal direction in the left posture to a more frontal one in the right posture.

Later investigators[2-8] found just the opposite, although most retained the same explanation. There is general agreement that lying on the left side usually deviates the mean frontal vector to the right (decreased R/S ratio in Lead I); lying on the right side deviates it usually to the left (increased R/S ratio). The former effect is more pronounced and more consistent than the latter.

None of the several theories proposed to explain this has ever been demonstrated adequately. Einthoven's explanation has been the favorite one. It was supported by the finding[15] that simple rotation of the heart on its anteroposterior axis in the dog produced much less change in the manifest vector than did rotation on the heart's long axis. Lewis[9] found in a patient with a bullet in the heart that a small anatomic shift, as indicated by the movement of the bullet roentgenologically, caused a large vector shift. Thus, it was not necessary to correlate the magnitudes of the electrical and anatomic changes. Other factors postulated were rotation of the heart about a sagittal axis[4] and changes of the heart's contact with the neighboring organs so that the conductivity to the electrodes changed.[4,5]

The little we know of the anatomic effects of postural change is based mostly on roentgenologic evidence.[14] In the lateral posture, gravity forces the mediastinum to the dependent side; the dependent diaphragm moves headward and the upper diaphragm footward. In the right lateral posture, the heart moves as a whole

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to the right. The left border becomes flatter and the right more arched, allegedly because the heart rotates about a caval axis, swinging like a door on the hinges formed by the caval veins. In the left lateral position the whole heart moves to the left; the apex is pushed upward by the rising left diaphragm. The rotation about the caval axis tilts the apex up even more and the left border more to the left. The heart's shape in this posture is decidedly transverse. The lateral motion of the heart ranges usually from 1.5 to 3 cm., sometimes to as much as 5 cm.

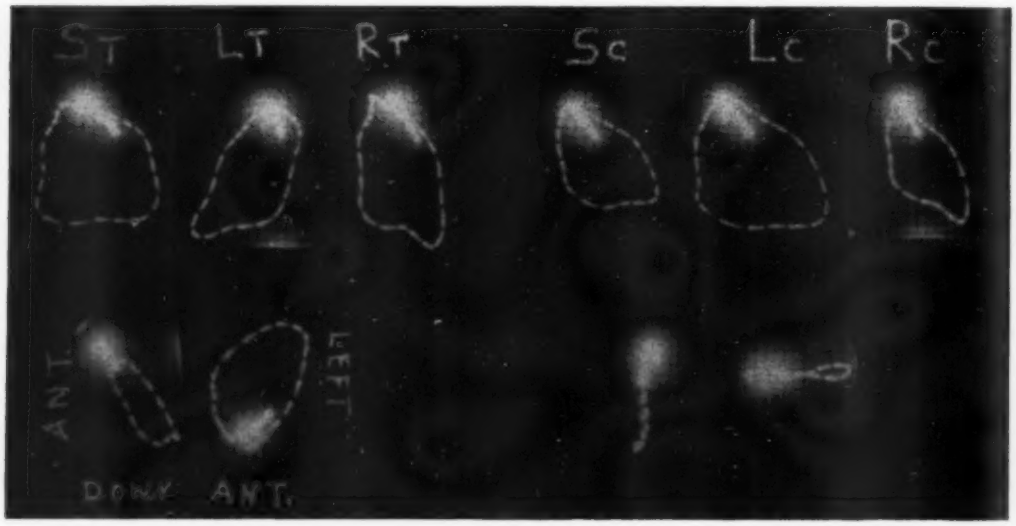



**Fig. 1.—E.C., 10 years old. Coarctation of aorta. Correspondence best between *RT* and *RC*, worse between *LT* and *LC*. Marked clockwise shift of *LT* while *LC* shows a mild counterclockwise shift. For all figures: Beam interrupted 400 times per second; blunt end of dash leading. The upper row shows the frontal (xy) plane in various postures. The lower row shows supine zy and xz planes. *S*, *L*, and *R* indicate supine, left lateral and right lateral postures. *T* and *C* indicate the tetrahedral and cube manifestations.**

Simple palpation reveals that in the left lateral posture the heart moves anteriorly as well as to the left and thus makes more intimate contact with the left chest wall.

Rotation about the Z (anteroposterior) axis takes place when the diaphragm ascends (expiration, pregnancy, ascites) and descends (inspiration, emphysema). The clockwise rotation of the heart in the frontal plane on descent of the diaphragm makes the heart more centric, while ascent makes it more eccentric. Rotation about other axes is a vague concept which is still in need of definition. Nor do we know any details of any possible rotation about a caval axis. It is anything but simple in view of the restraints imposed by the pulmonary veins, the pulmonary artery, and the aorta.

The effects of respiration on the frontal QRS vector are clearly parallel to the variations of the anatomic position of the heart. Descent of the diaphragm in inspiration rotates the heart and the vector clockwise; ascent in expiration rotates the two counterclockwise. On the other hand, the lateral postures cause changes in the manifest vector which cannot be correlated so simply with the anatomic changes.

Recently, we have learned to understand better the nature of the body as a volume conductor. Burger[11] showed how to handle the realities of the volume conductor in a practical way. It is now possible to take into account in the analysis of the manifest vector such factors as eccentricity, inhomogeneous conductivity, and irregular shape of the body. This investigation was undertaken because the new concepts appeared to explain the behavior of the manifest vector in the lateral postures.[12]

To understand the methods used in this investigation it is necessary to refer to Reference 13 for an explanation of the concepts of effective axis, image point of an electrode, and deviation of the manifest vector as a function of the deviation of the effective axis.

The fact that the left lateral posture usually deviates the manifest frontal vector to the right while the right lateral posture deviates it to the left seemed to be explainable by the new methods for analyzing the realities of the volume conductor. Thus, a shift of the heart as a whole to the left should deviate the effective axis of Lead I counterclockwise and thereby cause the manifest vector to deviate clockwise, and vice versa for the shift of the heart to the right. However, when the cube leads are used, then the effect should be the opposite.[12,13] This work was undertaken to test this hypothesis.

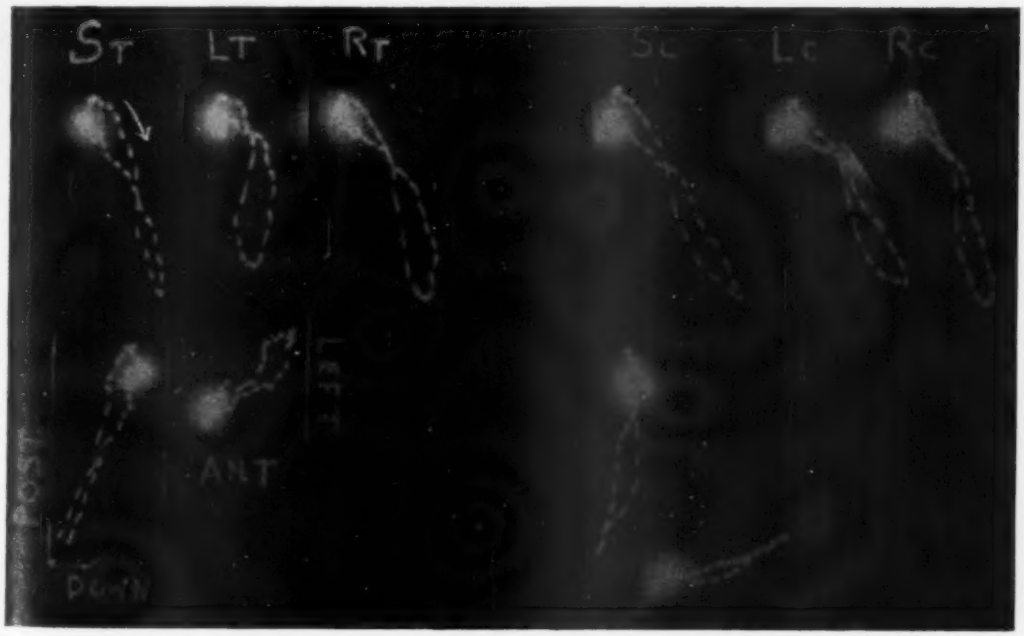



Fig. 2.—Be., 59 years old. Anteroinferior infarction. The efferent segment lies more posteriorly; it moves to right of the afferent segment in *LT*.

## TECHNIQUE

Vector- and electrocardiograms were recorded with the tetrahedral and cube lead systems.[17] The tetrahedral X, Y, and Z leads are I, $aV_F$ (or $V_F$) and $V_8$, respectively. The electrodes of the cube system are placed as follows: number 1 in the right posterior axillary line at the level of

the second lumbar vertebra; number 2 at the same level in the left posterior axillary line; number 3 at the same level in the right anterior axillary line; and number 4 over the right scapula in the posterior axillary line. The X, Y, and Z leads are A (2 minus 1), C (1 minus 4), and B (3 minus 1).

In a given system all leads had the same amplification which remained constant throughout a given experiment. Corrections to equalize the lengths of the effective axis would have been unrealistic because of our lack of quantitative data and would have complicated the procedure without improving the results.

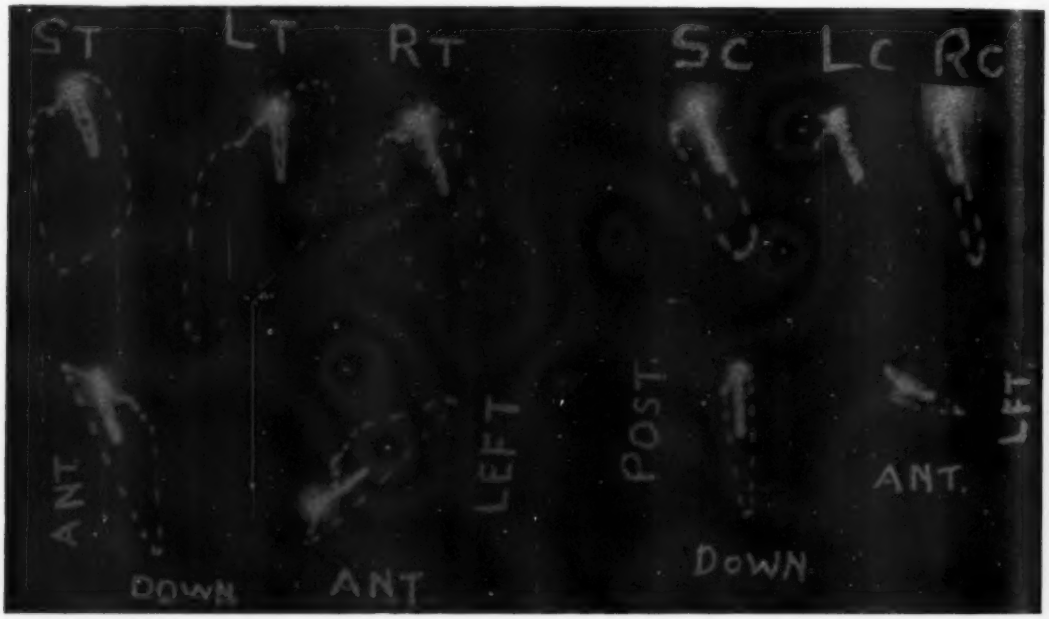



**Fig. 3.—S.G., 7 years old. Intraventricular septal defect probable. T loop shows postural changes.**

The frontal, sagittal, and horizontal planar loops were photographed in each position and with each lead system. Corresponding electrocardiograms of the component leads were also recorded. The record in the supine position was used as the reference state. The planar loops in the supine position as recorded by the tetrahedral system were used to form the wire spatial loop. The wire loops were manipulated to determine how they might reveal the planar loops manifested by the cube leads in the supine posture and by both systems in the various postures. The manipulation needed for this indicated the relative deviations of the effective axes of the leads.[12,13]

In the first group of 25 subjects recordings were made with both lead systems, in the supine, left lateral, and right lateral postures. In the second group of 11 subjects recordings were made with only the tetrahedral system, but in the prone position as well as in the other three postures.

In the first group 3 had a dextrocardia of which one was an instance of situs inversus with right bundle branch block; 1 was a typical example of Kartagener's syndrome (situs inversus, sinusitis, and bronchiectasis); the third had a dextroposition caused by a left diaphragmatic hernia, and the apex beat was felt at the tip of the xiphoid. In 2 patients the observations included ventricular extrasystolic beats in addition to the normal sinus beat. Six patients had left bundle branch block; 3 had right bundle branch block; 2 had myocardial infarction; and 4 showed right ventricular preponderance. Only 7 patients had a normal cardiovascular system. Of the 11 subjects in the second group, 2 had rheumatic mitral disease, 1 cor pulmonale, and 3 had mild heart disease of undetermined nature. There was no instance of marked cardiac enlargement, bundle branch block, or ventricular extrasystoles.

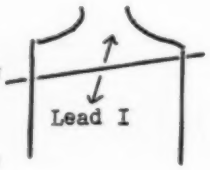
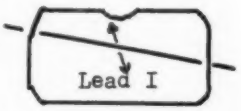
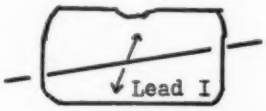
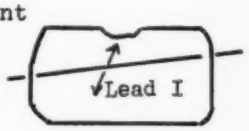
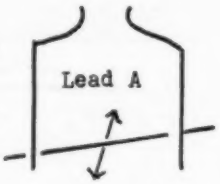
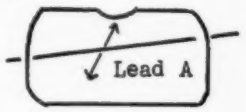
## RESULTS

In this report, the changes of the frontal (XY) planar loop only will be described, in order to avoid undue complexity. Three types of rotation were noted: in the frontal plane about the Z coordinate, in the horizontal plane about the Y coordinate, and in the sagittal plane about the X coordinate. The rotation about the Z coordinate is readily ascertained by estimating the angle alpha. In the other planes, the rotation can be deduced either from an inspection and manipulation of the spatial wire model or from the manner in which the efferent and afferent segments of the frontal planar loop cross and uncross.

**Fig. 4.—Ro., Left bundle branch block. Axis of Lead I in *LT* shows marked deviation around Y coordinate.**

**Fig. 5.—Mo., 70 years old. Left bundle branch block. Reversal of figure-of-eight in *LT* can be explained only by horizontal rotation of axis of Lead I about the Y coordinate.**

Table I. Results in Tabular and Diagrammatic Form

| Posture / Lead system / Type of case | Rotation about the Z coordinate (Frontal plane) No. | dVm | D.eff.ax. | Rotation about the Y coordinate (Horizontal plane) No. | dVm. | D.eff.ax. |
|---|---|---|---|---|---|---|
| Left lateral | | | | | | |
| Tetrahedral | | | | | | |
| Levocardia | 28/33 | cw |   | 25/33 | ccw |   |
| Dextrocardia | 2/3 | cw | | 2/3 | cw |   |
| Cube | No consistent deviation in any plane | | | | | |
| Right lateral | | | | | | |
| Tetrahedral | | | | | | |
| Levocardia | 23/33 | ccw | | No consistent effect | |   |
| Dextrocardia | 3/3 | ccw | | 3/3 | cw | |
| Cube | | | | | | |
| Levocardia | 14/22 | cw |   | No consistent effect | |   |
| Dextrocardia | 2/3 | cw | | 2/3 | cw | |
| Prone | | | | Rotation about the X coordinate (Sagittal plane) | | |
| Tetrahedral | | | | | | |
| Levocardia | 5/11 | ccw | | 6/11 | cw | |

*No.* shows the number of cases which showed the stated findings over the number examined. *dVm* indicates the direction of deviation of the manifest frontal vector; *cw*, clockwise; *ccw*, counterclockwise. *D.eff.ax.* shows a diagram of the deviation of the effective axis of the X or Y leads as a function of the deviation of the manifest vector according to the rule: A deviation of the effective axis clockwise deviates the manifest vector counterclockwise and vice versa. The tilt of the lead line indicates the direction of the deviation of the effective axis caused by posture, assuming that the lead axis in the supine posture is truly horizontal for Lead I and vertical for $V_F$. The tilt of the arrow indicates the direction of the deviation of the manifest vector, assuming that in the supine posture it was perpendicular to the lead axis.

The results are set forth in Table I. In the column labelled *eff. ax.*, all the deviations of the frontal vector are translated into deviations of the effective axis of Lead I (or in the case of the prone posture, of $V_F$). This was done to get the maximum information from the methods used here. Some of the postural effects noted here are probably not related to a change of the effective axes of the recording leads.

The ST-T loops showed similar changes when they were distinct enough to be analyzed (Figs. 3 and 6).

In many instances the loops of the two systems were most alike in the right lateral posture which minimizes the eccentricity (Figs. 1, 2, and 3). Again, the dextrocardia in Fig. 8 shows the reversed pattern in that the left lateral posture showed the most similar loops. These findings indicate clearly that the lateral shift of the heart is electrically very significant. They support the impression[12] that eccentricity is an important determinant of the differences between the manifestations of the two systems. Note that in the subject with the dextroposition of the heart the supine posture shows the best agreement between the two systems (Fig. 9). This indicates that the heart was most centric in this position.

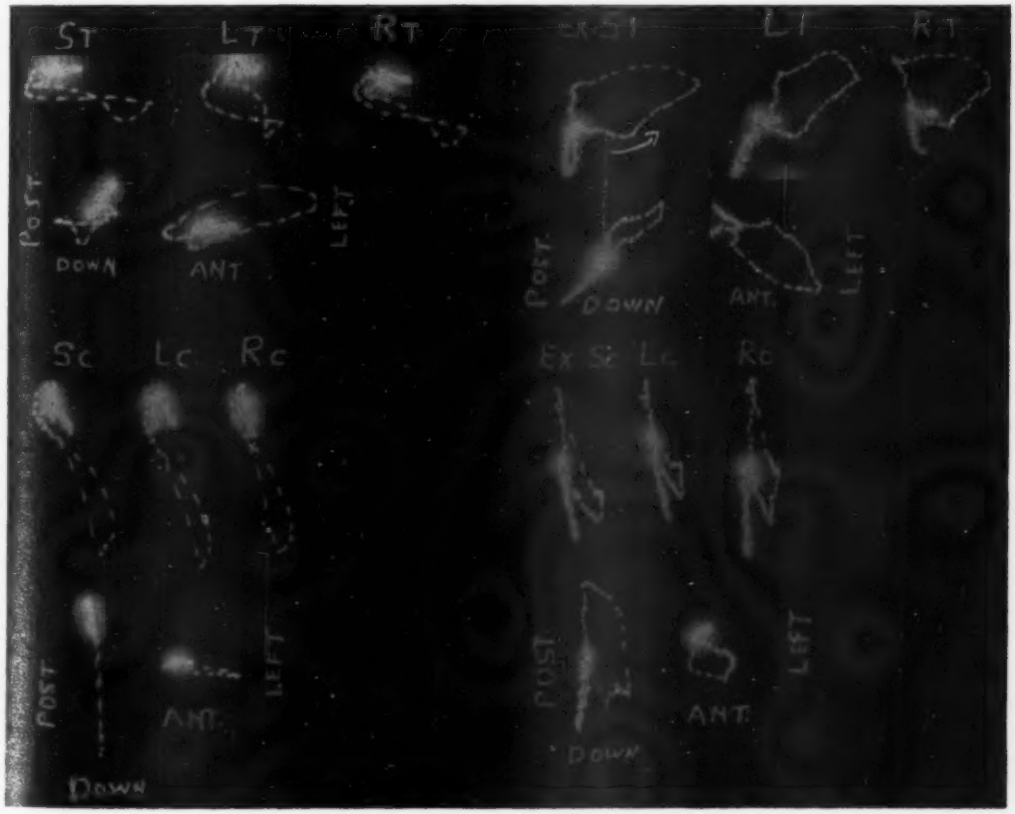



Fig. 6.—Cl., 13 years old. Ventricular extrasystoles. Normal beat on right; extrasystole (*Ex*) on left. See text.

## DISCUSSION

The results with respect to the tetrahedral loop are in accord with most of the previous reports. The left lateral posture usually decreases the R/S ratio in Lead I and deviates the frontal vector to the right. The right lateral posture usually increases the ratio and deviates the vector to the left.[2-8] Only Einthoven[1] reported an opposite pattern.

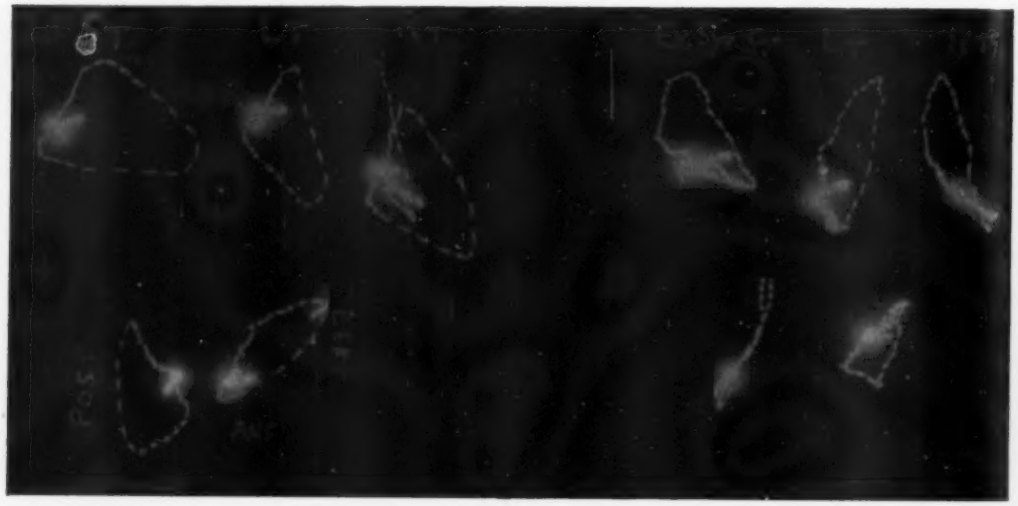


**Fig. 7.—M.C. Ventricular extrasystoles. Tetrahedral loops only. Postural changes more marked for extrasystole than for normal beat because deviation of manifest vector by rotating lead axis is a function of orientation of vector.**

The manifest vector would change in both lead systems in a like manner if the postural effects were caused by a rotation of the heart. The contrary proved to be the case. In the same posture the tetrahedral vector deviated one way and the cube the other way, or not at all. It is more likely that such postural effects are caused by a change within the volume conductor and not by a rotation of the heart. A practical method for analyzing volume conductor changes employs effective axes of the leads as a common denominator. Changes of the manifest vector are made a function of the lead axes which in turn are made a function of the changes in the volume conductor.

A deviation of the manifest vector clockwise from the control state signifies a deviation of the effective axis counterclockwise and vice versa. A deviation of the axis, in turn, is synonymous with a change of the image points of the electrodes and indicates a change in the conductivity between the heart and the electrodes (distance, tissue resistance, etc.).

The results indicated that the lateral postures affect chiefly the axes of the X lead, while the prone posture affects chiefly the Y lead. So, to avoid undue complexity and to arrive at a first approximation solution, only the X lead will be treated as a variable in the lateral posture and the Y lead in the prone posture.

The supine posture constitutes the control state. Changes are described

relative to the effective axes of the supine X and Y leads. Since the method is not quantitative, the results indicate chiefly directional changes.

Accordingly, postural changes can be translated as follows (Table I): The left lateral posture rotates the axis of Lead I counterclockwise in the frontal plane and clockwise in the horizontal. This posture has no consistent effect on the effective axis of Lead A. The right lateral posture rotates the axis of Lead I clockwise and that of Lead A counterclockwise in the frontal plane. The right posture has no consistent effect on these axes in the horizontal plane. The prone posture deviates the effective axis of $V_F$ clockwise in the frontal plane and counterclockwise in the sagittal plane (the superior tip moves dorsad and the inferior tip ventrad).

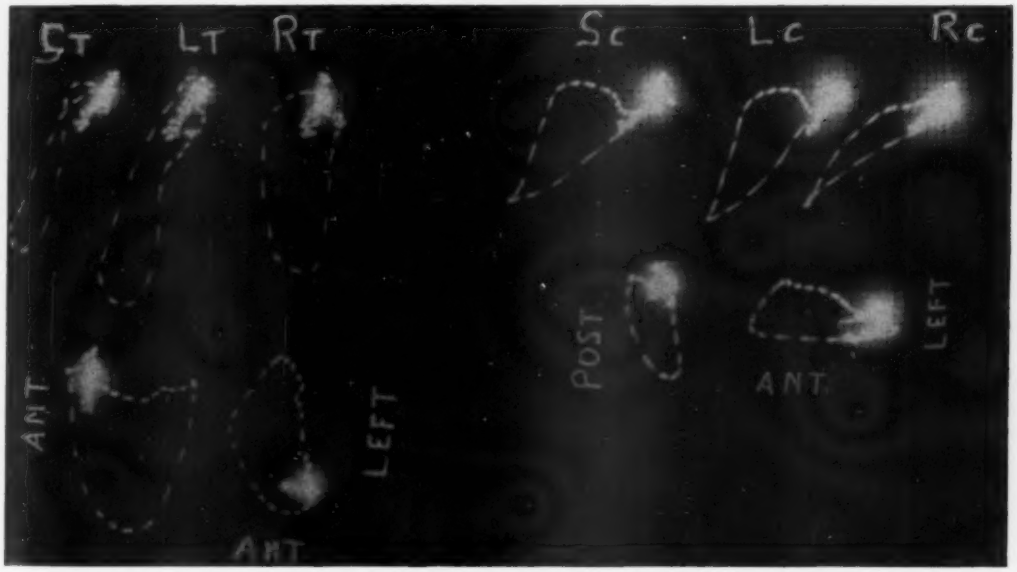



**Fig. 8.—L.B. Complete situs inversus with sinusitis and bronchiectasis. Recorded with $V_S$,R, and left-sided Leads B and C. *RT* shows maximal uncrossing of the loop in accord with a counterclockwise rotation of Lead I horizontally. The efferent segment lies inferiorly with a negligible sagittal component. It behaves in a manner consistent with eccentricity of heart to the right. In *ST* it lies counterclockwise from *SC*. It tends to be parallel to *LT* and *LC;* it is most divergent in *RT* and *RC*.**

The deviation of the effective axes of Lead I in the frontal plane is readily explained by the gravitational shift of the heart or blood, or of both. The heart moves as a whole and shortens the distance to the dependent electrode. The blood also shifts to the dependent side. Since it is a better conductor than the lung, the conductivity between the heart and the dependent electrode is improved. The shift of the blood may explain the marked postural change of the manifest vector which is found is some patients with constrictive pericarditis.

The shift of the heart and blood thus moves the image points of the dependent electrode away from the heart, and those of the superior electrode toward the heart. Thus, the effective axis of Lead I deviates in one direction while that of Lead A deviates in the other (Fig. 3).

The rotation of the axis of Lead I clockwise in the horizontal plane in the left lateral posture is as yet unexplained. If the rotation of the heart were the cause it would imply that the right ventricle rotates to the dependent side and the left ventricle to the upper side. Anatomically this appears unlikely. Furthermore, the cube leads did not reveal a similar rotation. It is conceivable that the left lateral posture approximates the heart more closely with the chest wall and thereby alters the image point of the left arm electrode. To test this hypothesis the second group of patients was studied in the prone as well as in the lateral postures. The results do not support this explanation for there is no consistent effect on the axis of Lead I, although the contact between heart and chest is greatest in the prone posture.

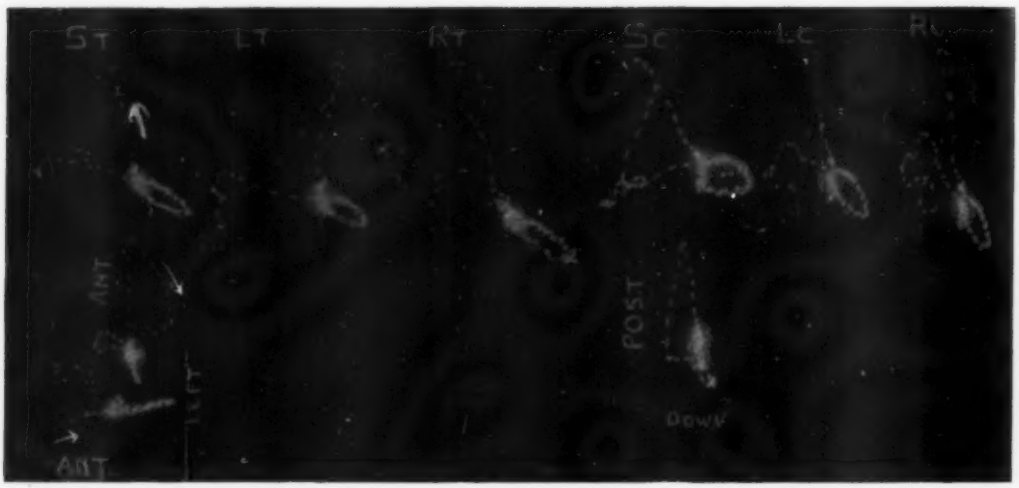



**Fig. 9.—Ma. Dextroposition of heart caused by left diaphragmatic hernia. Right bundle branch block. *ST* and *SC* most alike as if heart is centric in supine position. *RT* shows maximal horizontal deviation of axis of Lead I counterclockwise, in that the posterior segment moves to the left and the anterior parts (especially terminal slow complex) moves to right. Analysis with wire spatial loop especially informative.**

The dextrocardias support the concept that the shift of the heart toward the dependent side deviates the effective axis of the X leads by moving the image point of the dependent arm electrode away from the heart. Note that in the dextrocardias the right lateral posture has a stronger effect than the left in deviating the frontal planar tetrahedral loop in the horizontal plane. Note also that here the anterior segment of the loop moved toward the dependent side (Fig. 8), similar to the levocardias.

It is not clear how the prone posture brings about the changes in the manifest vector. The best explanation for the counterclockwise deviation in the frontal plane seems to be an anatomic one and not related to a change in the effective axes of the leads. The prone position probably makes the heart more transverse by causing the diaphragm to rise. The deviation in the sagittal plane, whereby the dorsal segment of the loop moves superiorly compared to the anterior segment, is without apparent explanation.

The cube frontal loop showed no consistent change in the left lateral posture and showed less change than the tetrahedral loop in the right lateral posture. In part this may be explained by the nature of the electrodes of Lead A as compared to those of Lead I. The potential gradient of the shoulders is steep as compared to that of the mid-back. This is well illustrated in a recent report by Frank,[10] where the potential gradient is expressed as a density of the isopotential lines. A given change in the volume conductor has a greater effect on an electrode located in the area with a steep potential gradient than on an electrode in a low gradient region.[16] Accordingly, the image points of Lead I would vary much more than those of Lead A for a given shift of the heart.

It is not clear why the left lateral posture has such a small effect on the cube manifestation.

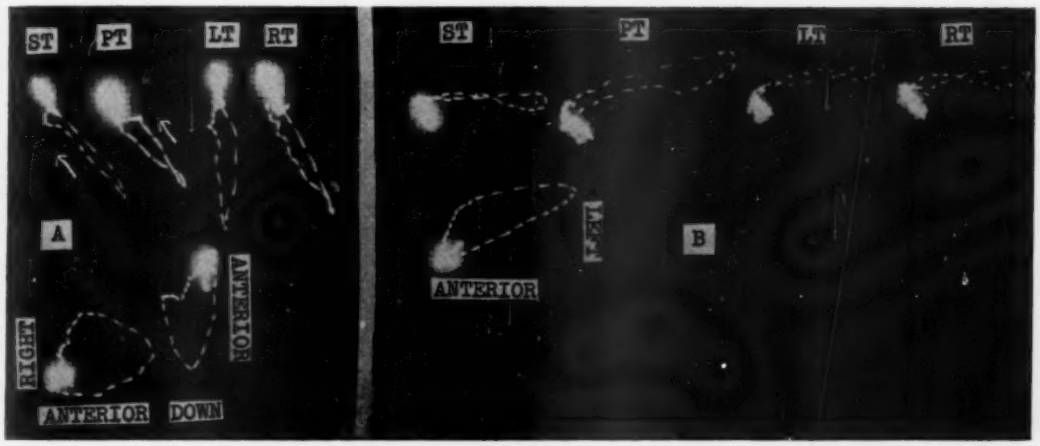



**Fig. 10.—*PT* is the tetrahedral frontal loop recorded in the prone posture. A.Gr: *ST* frontal loop is inscribed clockwise; *PT* counterclockwise. B.Or: *ST* frontal loop inscribes figure-of-eight; *PT* counterclockwise. In both, the prone posture has moved the posterior segment of loop superiorly of the anterior; equivalent to a counterclockwise deviation of effective axis of $V_F$ about the X coordinate.**

In Fig. 6, the correspondence between ST and SC especially for the Y dimension is much worse for the normal beat than for the extrasystolic beat. It can be explained in part by assuming that the upper end of YT (effective axis of Lead $V_F$) lies to the left and posterior of that of YC (effective axis of Lead C). To the extent that the heart vector has a left and posterior component, SC will lie inferior to ST. The normal beat has the QRS loop situated to the left and posterior, and, accordingly, SC here has a large inferior position while ST has a small inferior component. When the vector has an anterior component, SC will lie superior to ST. Since the extrasystolic QRS lies to the left and anterior, it will be subject to both inferior and superior deviating forces. The net effect in SC will be a decreased deviation in SC, and the correspondence between SC and ST will be improved.

The variable correspondence of normal and extrasystolic beats has been noted by others and has been explained by assuming a change in the position of the heart dipole for different beats.[18] (See a previous report for a discussion of this

subject.[13]) The author believes that much of the variable correspondence can be explained as a function of the orientation of the heart vector. Likewise, the difference in the postural effects on the normal and the extrasystolic beat (Figs. 6 and 7) is also a function of the orientation of the heart vectors. It is based on the rule[12] that the deviation of the manifest vector by deviating lead axes is greatest for those vectors perpendicular to the axes and minimal for those parallel to the axes.

## SUMMARY

The effects of the left and right lateral postures on the tetrahedral and cube frontal planar vectorcardiograms were studied in 25 patients of whom 2 had total situs inversus and one an acquired dextroposition. In a second group of 11 patients the effects of the prone as well as of the lateral postures were studied, but only with respect to the tetrahedral frontal loop.

The results indicated that the anatomic rotation of the heart about its various axes cannot account for most of the changes. Much can be explained by a shift of the heart as a whole toward the dependent side. This alters the characteristics of the body as a volume conductor, which may be expressed as changes of the effective axes of the leads especially the X leads. In turn, these change the manifest vector. The image point of the left arm appears to be especially sensitive to postural effects. Some of the results are as yet unexplained.

The manifest vectors of extrasystoles showed a different amount of postural deviation than did that of normal beats in the same individual. This could be explained readily by the rule that the deviation of the manifest vector by deviating lead axes is greatest for those vectors oriented perpendicularly to the axes and minimal for those parallel to the axes.

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# PHYSICAL BASIS OF BALLISTOCARDIOGRAPHY. V

## The Distortion of the Ballistocardiogram Caused by the Movement of the Heart Inside the Body

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### INTRODUCTION

THE problem of the difference in movement of a ballistocardiograph (bcg) and the subject lying on it has been treated quantitatively for various types of bcg's.[1,2] A quantitative treatment of the difference in movement of the heart and a part of its surrounding tissues with respect to the skeleton has not yet been given. In the present paper the latter treatment will be given while taking the former into account. The necessary data, concerning the binding of the heart to the skeleton, will be taken from the literature.[3]

### THEORY

The problem that will be treated in this paper is a generalization of that in which only the difference in movement of subject and bcg has been considered. The latter has been schematized as follows[1,2,4]: The subject and the bcg are coupled by a directive force and a frictional force. The bcg itself is coupled not only to the subject but also to the surroundings. The latter coupling is also represented by a directive force and a frictional one.

In the above intended schema the subject is represented by a single mass. In reality, the body must be represented by a great many masses in the three-dimensional space, all coupled mutually. It has been shown that the binding between the larger outer parts, namely, the legs and the head, is, as a first approximation, strong enough to be assumed infinitely strong.[2]

A very important coupling is that of the heart to the skeleton.[2,3,4,8,9] The coupling appears not to be strong,[3] so that they can move independently to some extent. As the ballistocardiographic effect is caused by the action of the heart, it will be of importance to realize the influence of the weakness of this binding upon the ballistocardiogram (BCG). As a result of this weak binding, the heart, with a part of the tissues surrounding it, will move with respect to the rest of the body. The former will be referred to as the "heart," the latter as the "subject." The "heart" will not move as a single mass. It is a problem of a movement

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within an almost continuous medium. However, to make the problem at issue solvable, the "heart" is assumed to move as a single mass as is done for the "subject."

The more general schema in which the binding between the "heart" with mass $m_h$ and the "subject" with mass $m'_s$, as well as that between the "subject" and the bcg with mass $m_b$, and that between the bcg and the surroundings are represented as shown in Fig. 1,*A*. Fig. 1,*B* gives a somewhat simpler illustration of the same system, all composing parts being drawn beside each other. This way of schematization has been proposed already,[4] just like the couplings, each of them being built up from a directive force and a frictional one.

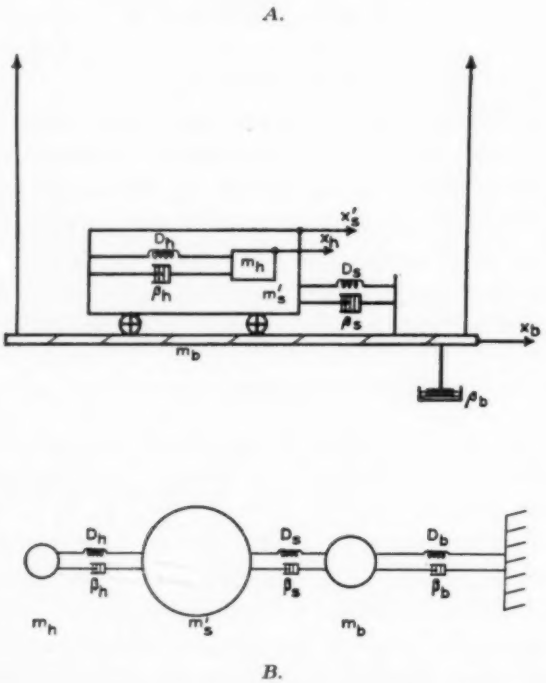


**Fig. 1.—*A*, Schematic representation of the ballistocardiographic system. The "heart" of the subject is coupled to the subject by a directive force and a frictional one. The couplings between subject and bcg and between bcg and surroundings are assumed as being built up in the same way. The masses of the "heart," the "subject," and the bcg are marked by $m_h$, $m'_s$, and $m_b$, respectively. The different couplings are represented schematically. *B*, A simpler illust ation of the system of *A*.**

To find the influence of the various couplings upon the behavior of the different parts, it is necessary to write down the differential equations in which the forces acting on the different parts of the system are included. The way in which these equations are to be found has been described in previous papers[2,5] and will not be repeated here. In the case that there are two masses movable with respect to each other, two simultaneous differential equations have been found.[2] In this case there are three masses movable with respect to each other. Then three simultaneous differential equations describe the movement.

The driving force is assumed to work only inside the "heart." It equals the product of the body mass $m_e$ and the acceleration of the center of gravity $\ddot{x}_c$.

The displacement $x_c$, the velocity $\dot{x}_c$ and the acceleration $\ddot{x}_c$ of the center of gravity of the subject with mass $m_s$ ($m_s = m'_s + m_h$) are reckoned with respect to the skeleton and chosen positive to the left in Fig. 1 (footward). The respective quantities for the "heart" (index h), the "subject" (index s), and the bcg (index b) are calculated with respect to the surroundings and chosen positive to the right (headward). Every frictional force acting between two masses is assumed to be proportional to the difference in velocity of those two masses. The proportion factors are called $\beta$ and provided by corresponding indexes as is shown in Fig. 1. In an analogous way every directive force is assumed to be proportional to the difference in displacement of the masses. The proportion factors are called D and are indexed in the same way. The three differential equations holding for this system then read as follows:

$$m_s\ddot{x}_c - \beta_h(\dot{x}_h - \dot{x}'_s) - D_h(x_h - x'_s) = m_h\ddot{x}_h \tag{1}$$

$$\beta_h(\dot{x}_h - \dot{x}'_s) + D_h(x_h - x'_s) - \beta_s(\dot{x}'_s - \dot{x}_b) - D_s(x'_s - x_b) = m'_s\ddot{x}'_s \tag{2}$$

$$\beta_s(\dot{x}'_s - \dot{x}_b) + D_s(x'_s - x_b) - \beta_b\dot{x}_b - D_b x_b = m_b\ddot{x}_b \tag{3}$$

In the case that the binding between the "heart" and the "subject" is infinitely strong ($D_h = \infty$) both the differential equations holding for the problem that there is only difference in movement between subject and bcg are found again.[2]

The center of gravity of the subject is assumed to move periodically and, hence, can be developed in a Fourier series. A term arbitrarily chosen from this series can be written in the usual exponential form

$$x_c = |x_c|\, e^{j\omega t} \tag{4a}$$

in which $x_c$ is the displacement and $|x_c|$ is the amplitude of this displacement of the center of gravity, $\omega = 2\pi\nu$, $\nu$ the frequency and $j^2 = -1$.

The solution of the differential equations (1), (2), and (3), valid if the movement of the various masses is stationary, can be represented by

$$x_h = |x_h|\, e^{j(\omega t + \varphi_h)} \tag{4b}$$

$$x'_s = |x'_s|\, e^{j(\omega t + \varphi'_s)} \tag{4c}$$

$$x_b = |x_b|\, e^{j(\omega t + \varphi_b)} \tag{4d}$$

$|x_h|$, $|x'_s|$ and $|x_b|$ represent the amplitude of the displacement of the "heart," the "subject," and the bcg, respectively. But their values must still be calculated. The phase shift (time-lag) between the mass movement within the subject and between the movement of the "heart," the "subject," and the bcg, respectively, is indicated by $\varphi_h$, $\varphi'_s$, and $\varphi_b$.

By substituting the formulas (4a), (4b), (4c), and (4d) in the three differential equations, the amplitude distortion and the time-lag can be derived by a simple but somewhat elaborate calculation. Moreover, the difference in movement of two arbitrarily chosen masses follows from this calculation. It gives

$a$ for the amplitude of the "heart"

$$|x_h| = m_s \omega^2 L_h |x_e| \tag{5}$$

$b$ for the amplitude of the "subject"

$$|x_s| = m_s \omega^2 L_s |x_e| \tag{6}$$

$c$ for the amplitude of the bcg

$$|x_b| = m_s \omega^2 L_b |x_e| \tag{7}$$

The abbreviations $L_h$, $L_s$, and $L_b$ stand for:

$$L_h = \left[\frac{h_1^2 + h_2^2}{n_1^2 + n_2^2}\right]^{\frac{1}{2}}, \quad L_s = \left[\frac{s_1^2 + s_2^2}{n_1^2 + n_2^2}\right]^{\frac{1}{2}}, \quad L_b = \left[\frac{b_1^2 + b_2^2}{n_1^2 + n_2^2}\right]^{\frac{1}{2}} \tag{5a), (6a), (7a}$$

in which

$$h_1 = -\omega^4 m'_s m_b + \omega^2 (m'_s D_s + m'_s D_b + m_b D_h + m_b D_s + \beta_h \beta_s + \beta_h \beta_b + \beta_s \beta_b) - (D_h D_s + D_h D_b + D_s D_b)$$

$$h_2 = \omega^3 (m'_s \beta_s + m'_s \beta_b + m_b \beta_h + m_b \beta_s) - \omega(\beta_h D_s + \beta_h D_b + \beta_s D_h + \beta_s D_b + \beta_b D_h + \beta_b D_s)$$

$$s_1 = \omega^2 (m_b D_h + \beta_h \beta_s + \beta_h \beta_b) - (D_h D_s + D_h D_b)$$

$$s_2 = \omega^3 m_b \beta_h - \omega(\beta_h D_s + \beta_h D_b + \beta_s D_h + \beta_b D_h)$$

$$b_1 = \omega^2 \beta_h \beta_s - D_h D_s$$

$$b_2 = -\omega(\beta_h D_s + \beta_s D_h)$$

$$n_1 = -\omega^6 (m_h m'_s m_b) +$$
$$+\omega^4 (m_s m_b D_h + m_h m_b D_s + m_h m'_s D_s + m_h m'_s D_b + m_h \beta_s \beta_b + m_s \beta_h \beta_b + m_s \beta_h \beta_s + m_b \beta_h \beta_s) -$$
$$-\omega^2 (m_h D_s D_b + m_s D_h D_b + m_b D_h D_s + m_s D_h D_s + \beta_h \beta_s D_b + \beta_h \beta_b D_s + \beta_s \beta_b D_h) + (D_h D_s D_b)$$

$$n_2 = \omega^5 (m_h m'_s \beta_s + m_h m'_s \beta_b + m_h m_b \beta_s + m_s m_b \beta_h) -$$
$$-\omega^3 (m_h \beta_b D_s + m_h \beta_s D_b + m_s \beta_h D_b + m_s \beta_s D_h + m_s \beta_b D_h + m_s \beta_h D_s + m_b \beta_h D_s + m_b \beta_s D_h + \beta_h \beta_s \beta_b)$$
$$+\omega(\beta_h D_s D_b + \beta_s D_h D_b + \beta_b D_h D_s)$$

$d$ for the ratio between the amplitude of the difference in displacement of "heart" and "subject" and the amplitude of the displacement of the "heart"

$$\left|\frac{x_h - x'_s}{x_h}\right| = \left[\frac{(h_1 - s_1)^2 + (h_2 - s_2)^2}{h_1^2 + h_2^2}\right]^{\frac{1}{2}} \tag{8}$$

We calculated this ratio because of the reason that it is interesting to know whether the difference in movement of "heart" and "subject" is strongly dependent on the type of bcg that is made use of. It would be obvious to calculate for that purpose the amplitude of the difference in displacement of "heart" and "subject" $|x_h - x'_s|$ as a function of frequency. However, it will be clear that this amplitude does not give a reliable answer to the question. For the displacements of both types of bcg's are widely different for the same subject. Consequently we took the ratio between the above-mentioned amplitude and the amplitude of the displacement of the "heart."

*e* for the phase shift (time-lag) between the displacement of the center of gravity and the displacement of the "heart" $\varphi_h$

$$tg\varphi_h = \frac{h_2 n_1 - h_1 n_2}{h_1 n_1 + h_2 n_2} \tag{9}$$

*f* for the phase shift (time-lag) between the displacement of the center of gravity and the displacement of the "subject" $\varphi'_s$

$$tg\varphi'_s = \frac{s_2 n_1 - s_1 n_2}{s_1 n_1 + s_2 n_2} \tag{10}$$

*g* for the phase shift (time-lag) between the displacement of the center of gravity and the displacement of the bcg $\varphi_b$

$$tg\varphi_b = \frac{b_2 n_1 - b_1 n_2}{b_1 n_1 + b_2 n_2} \tag{11}$$

If the BCG is obtained by recording the displacement of a *low-frequency* bcg (natural frequency of the loaded instrument low with respect to the heart frequency) the recorded curve is intended to approximate the displacement of the center of gravity $x_c$. The above given formulas (5) to (11) can be applied on this type of bcg.

If the BCG is obtained by recording the displacement of a *high-frequency* bcg (natural frequency of the loaded bcg high with respect to the heart frequency) the recorded curve is intended to approximate the acceleration of the center of gravity $\ddot{x}_c$. So, in these formulas $\ddot{x}_c$ is to be considered as required, as $x_c$ was in the cases described above. For the latter type of bcg the formulas below hold, which follow from those given above by a simple transformation. It gives

*a* for the amplitude of the "heart"

$$|x_h| = m_s L_h |\ddot{x}_c| \tag{12}$$

*b* for the amplitude of the "subject"

$$|x_s| = m_s L_s |\ddot{x}_c| \tag{13}$$

*c* for the amplitude of the bcg

$$|x_b| = m_s L_b |\ddot{x}_c| \tag{14}$$

*d* for the ratio between the amplitude of the difference in displacement of "heart" and "subject" and the amplitude of the displacement of the "heart" formula (8)

*e* the phase shift (time-lag) between the acceleration of the center of gravity and the displacement the "heart," the "subject," and the bcg $\varphi_h^*$, $\varphi'^*_s$ and $\varphi_b^*$, respectively, are to be found from $\varphi_h$, $\varphi'_s$ and $\varphi_b$ (formulas (9), (10), and (11)) by a relation derived earlier.[5]

## RESULTS

To make the results of the calculations given in the formulas more easily accessible, the amplitude distortion and the phase shift can be represented graphically. For that purpose, it is necessary to know the numerical values of the constants that are met with in these formulas.

For the values of $m_s = m'_s + m_h$, $m_b$, $\beta_s$, $\beta_b$, $D_s$, and $D_b$, the same numbers will be used as in a preceding paper.[2] They are found in Table I. The improvements yielded by Starr[10] could not yet be taken into account.

TABLE I

| | LOW-FREQUENCY bcg (BURGER) | | | HIGH-FREQUENCY bcg (STARR) | | |
|---|---|---|---|---|---|---|
| | I | II | III | IV | V | VI |
| $m_h$ (Kg.) | 0.5 | 1.5 | 4.5 | 0.5 | 1.5 | 4.5 |
| $m'_s$ (Kg.) | 73.5 | 72.5 | 69.5 | 73.5 | 72.5 | 69.5 |
| $m_s$ (Kg.) | 74 | 74 | 74 | 74 | 74 | 74 |
| $m_b$ (Kg.) | 6.0 | 6.0 | 6.0 | 15 | 15 | 15 |
| $D_h$ ($10^3$Kg. s$^{-2}$) | 1.3 | 3.8 | 12 | 1.3 | 3.8 | 12 |
| $D_s$ ($10^4$Kg. s$^{-2}$) | 6.5 | 6.5 | 6.5 | 6.5 | 6.5 | 6.5 |
| $D_b$ ($10^2$Kg. s$^{-2}$) | 2.9 | 2.9 | 2.9 | $80.10^2$ | $80.10^2$ | $80.10^2$ |
| $\beta_h$ (Kg. s$^{-1}$) | 8.5 | 26 | 77 | 8.5 | 26 | 77 |
| $\beta_s$ ($10^3$Kg. s$^{-1}$) | 9.8 | 9.8 | 9.8 | 9.8 | 9.8 | 9.8 |
| $\beta_b$ (10 Kg. s$^{-1}$) | 12 | 12 | 12 | 9.6 | 9.6 | 9.6 |

**The numerical values of the constants appearing in the formulas given above which hold good for the low-frequency bcg with a (loaded or not loaded) natural frequency of 0.3 c/s and for the high-frequency bcg with a loaded natural frequency of 15 c/s. The values of the constants determining the binding between "subject" and bcg (with index s) are used in case a footplate is applied. Except those concerning the binding between "heart" and "subject," all constants are taken from an earlier paper.[2]**

It is more difficult to trace numerical data concerning $m_h$, $\beta_h$, and $D_h$. For $m_h$, the mass of the "heart," we have chosen three values, namely,

$$m_h = 0.5,\ 1.5, \text{ and } 4.5 \text{ Kg.}$$

These values have been chosen for two reasons. In the first place, to find out what will be the influence of the numerical value of the "heart" mass on the reliability of the recorded curve. In the second place, because we are convinced that this region includes the correct values of $m_h$ occurring in practice. Data concerning $D_h$ are given in the literature.[3,4] Von Wittern[4] gives for the natural frequency of the "heart" about 5 c/s, but that number might have been deduced from a single measurement. Grandell[3] gives as a result of a series of measurements, done in another way (though at necropsy), a mean value of 8 c/s. For a reason that will be mentioned below, we prefer the use of the latter number. With the aid of the approximating formula

$$4\pi^2\nu_h^2 = D_h/m_h,$$

holding for not too large dampings, the following data for $D_h$ are found, using the above given data of $m_h$ respectively:

$$D_h = 1.3\ 10^3,\ 3.8\ 10^3, \text{ and } 12\ 10^3 \text{Kg. s}^{-2}.$$

We found data concerning $\beta_h$ only in the above paper of Grandell. He gives for the experimentally determined damping about 65 per cent per cycle as a mean value. We interpreted that this meant (Fig. 2)

$$\frac{x_0 - x_2}{x_0} = 0.65.$$

From this the damping coefficient $\beta_h$ can be derived in the following way. As for a free vibration $x_1/x_0 = x_2/x_1$ holds, it follows for the amplitude ratio in two successive inversion points:

$$\frac{x_1}{x_0} = (1 - 0.65)\,\tfrac{1}{2} = 0.59.$$

From this ratio, making use of a formula deduced earlier,[6] it follows for the ratio $\delta_h$ of the damping $\beta_h$ and the critical damping $(\beta_h)crit$:

$$\delta_h = 0.17.$$

The critical damping equals: $(\beta_h)crit = 2\sqrt{m_h D_h}$, so $\beta_h$ can be calculated for the three above-mentioned cases in which the value of $\delta_h$ is kept constant. It results in:

$$\beta_h = 8.5,\ 26,\ 77\ Kg.\ s^{-1}.$$

These numbers complete the necessary numerical data. They are tabulated in Table I.

From the given formulas (5) to (11) all characteristics are to be found when use is made of the data given in Table I. But as the calculations are rather extensive, only a few representative characteristics have been worked out numerically. The following characteristics are represented:

1. The amplitude characteristics of the *low-frequency* bcg if the displacement of the center of gravity $x_c$ is found by recording the displacement $x_s'$ of the "subject." They are calculated for the cases that the mass of the "heart" equals 0.5, 1.5, and 4.5 Kg. (Fig. 3). For frequencies higher than 4 c/s the heights of these characteristics differ only a few percentages. Therefore, only one line could be drawn in this region. Also a phase characteristic holding for the case that $m_h = 1.5\ Kg.$ has been drawn as a representative for the three cases corresponding to the three values of $m_h$ (Fig. 4).

2. The amplitude characteristics of the *low-frequency* bcg if the displacement of the center of gravity $x_c$ is found by recording the displacement $x_b$ of the bcg. The mass of the "heart" equals 0.5, 1.5, and 4.5 Kg. (Fig. 5). For frequencies higher than 4 c/s the heights of the characteristics again differ only a few percentages. In the case of $m_h = 1.5\ Kg.$ the phase characteristic is represented too (Fig. 6).

3. The amplitude characteristic of the *high-frequency* bcg if the acceleration of the center of gravity $\ddot{x}_c$ is found by recording the displacement of the "subject" $x_s'$. The mass of the "heart" equals 1.5 Kg. (Fig. 7).

4. The amplitude characteristic of the *high-frequency* BCG if the acceleration of the center of gravity $\ddot{x}_c$ is found by recording the displacement of the bcg $x_b$. The mass of the "heart" equals 1.5 Kg. (Fig. 8).

5. The ratio between the amplitude of the difference in displacement of "heart" and "subject" $[x_h\text{-}x_s']$ and the amplitude of the displacement of the "heart" $|x_h|$, if a *low-frequency* bcg is used (Fig. 9, *lf*).

6. The ratio between the amplitude of the difference in displacement of "heart" and "subject" $|x_h\text{-}x_s'|$ and the amplitude of the displacement of the "heart" $|x_h|$ if a *high-frequency* bcg is used (Fig. 9, *hf*).

## DISCUSSION

*1.* The given formulas have been checked by the examination of extreme cases. For instance, for the case drawn schematically in Fig. 10, in which the binding between "heart" and "subject" is supposed to be extremely strong

($D_h$ and/or $\beta_h = \infty$), must hold the formula given for a more simple case.[2] Moreover, $|x_h|$ from formula (5) must then be equal to $|x'_s|$ from formula (6), for two rigidly coupled masses must move in the same way. In the same case, the phase shift $\varphi'_s$ in formula (10) must be equal to the phase calculated earlier.[2]

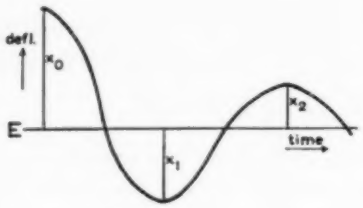


**Fig. 2.—The deflection from the equilibrium position $E$ of an unforced damped system that is brought out of that position plotted with respect to time.**

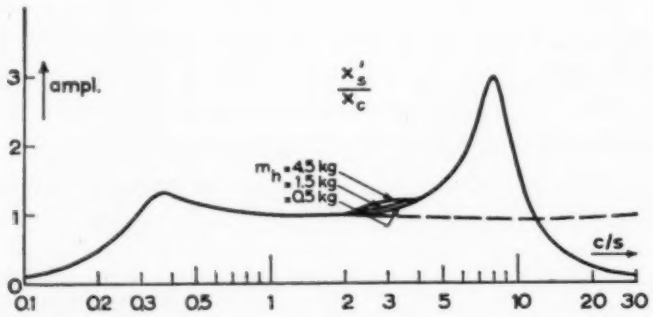


**Fig. 3.—The amplitude characteristics of a *low-frequency* bcg if the displacement of the center of gravity $x_c$ is found by recording the displacement of the "subject" $x'_s$. The difference between the characteristics is only more than a few percentages in the neighborhood of 3 c/s. The broken line holds if the binding between "heart" and "subject" were infinitely strong.**

In this way a number of other special cases are to be found. All checks gave the results that were predicted for the corresponding simpler cases.

*2.* In Figs. 3 and 5 a high resonance peak appears in the place where the frequency $\nu$ equals the natural frequency of the "heart" with respect to the fixed "subject" ($\nu = 8$ c/s). In a preceding paper,[2] in which only the relative movement of subject and bcg was taken into account, this high resonance peak did not occur. To find out when such a peak occurs and when it does not, the behavior of the system in two simplified cases will be represented graphically.

In both simplified cases the binding between "subject" and bcg is assumed to be infinitely strong ($D_s$ and/or $\beta_s = \infty$). Therefore, the masses inside the outlined area of Fig. 11, *A* move as a whole. Fig. 11, *B* gives a simpler drawing of the same system. The amplitude characteristic of this system is represented in Fig. 13, curve 1. It is assumed that the displacement of the center of gravity is found by recording the displacement of the mass $m'_s + m_b$. The second simplified system equals the first except that the masses $m_h$ and $m'_s + m_b$ have been

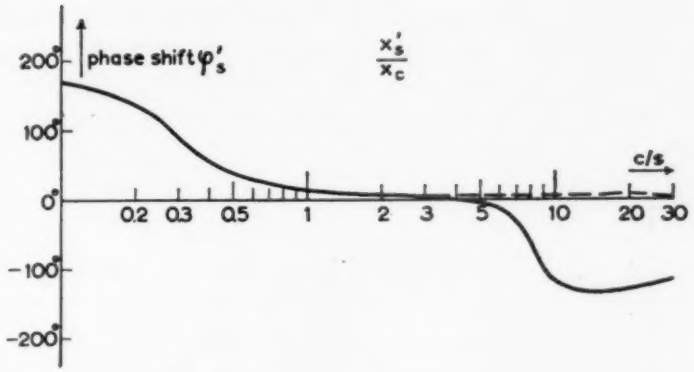


Fig. 4.—The phase characteristic of a *low-frequency* bcg if the displacement of the center of gravity $x_c$ is found by recording the displacement of the "subject" $x'_s$, belonging to a value of the mass of the "heart" of 1.5 Kg. The broken line gives the characteristic if there were no difference in movement between "heart" and "subject."

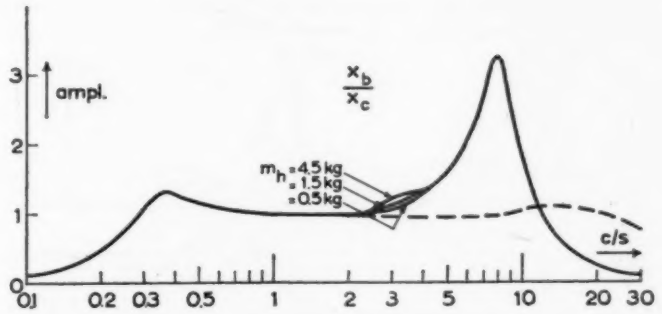


Fig. 5.—The amplitude characteristics of a *low-frequency* bcg if the displacement of the center of gravity $x_c$ is found by recording the displacement of the bcg $x_b$. The characteristic representing the case that "heart" and "subject" were infinitely strongly bound to each other is given by the broken line.

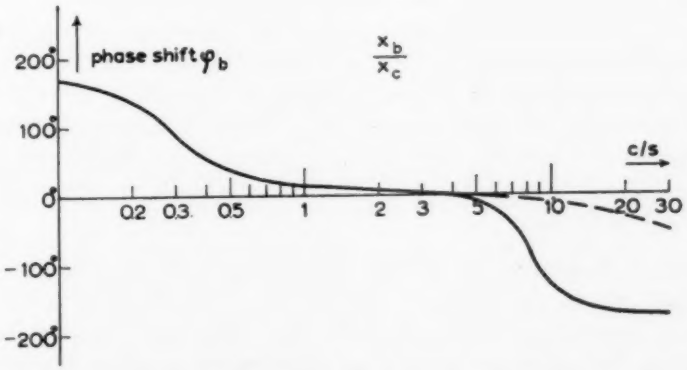


Fig. 6.—The phase characteristic of a *low-frequency* bcg if the displacement of the center of gravity $x_c$ is found by recording the displacement of the bcg $x_b$ belonging to a value of the mass of the "heart" of 1.5 Kg. The characteristic representing the case that the compliance of the "heart" would be zero is shown by the broken line.

interchanged (Fig. 12). The amplitude characteristic of this system is represented in Fig. 13, curve 2. The results are scarcely dependent on the choice of $m_h$. In this case it is assumed that the displacement of the center of gravity is found by recording the displacement of the mass $m_h$. In both cases the numerical values of Table I have been used.

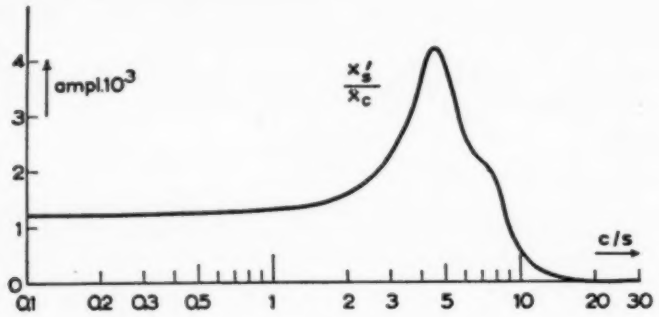


Fig. 7.—The amplitude characteristic of a *high-frequency* bcg if the acceleration of the center of gravity $\ddot{x}_c$ is found by recording the displacement of the "subject" $x'_s$. The mass of the" heart" has been chosen 1.5 Kg. The ordinates are given in square seconds.

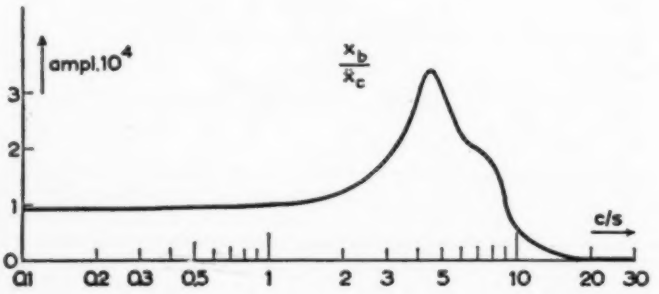


Fig. 8.—The amplitude characteristic of a *high-frequency* bcg if the acceleration of the center of gravity $\ddot{x}_c$ is found by recording the displacement of the bcg $x_b$. The ordinates are given in square seconds.

From the characteristics of Fig. 13 it appears that the height of the above-mentioned resonance peak is highly dependent on the sequence of the masses if the coupling constants remain constant. From this it is understandable that in the characteristics represented in Figs. 3 and 5 the height of the resonance peak may be great. Moreover, it is understandable why the resonance peak in an earlier paper[2] was low.

*3.* In the calculations to obtain the characteristics represented in Figs. 3 and 5 the mass $m_h$ of the "heart" equals 0.5, 1.5, and 4.5 Kg. Grandell[3] gives for the mass of the proper heart about 0.5 Kg. But not only the heart will move with respect to the skeleton; a part of the tissues surrounding it will be made to move too. The tissue close to the heart will perform the same movement as the heart, while the tissue will carry out a smaller movement as it is more removed from it. We introduced therefore an effective mass $m_h$. The radius of this

effective mass is assumed to be about 2 cm. larger than that of the heart. From this it follows for the value of $m_h$ about 1.5 Kg.

To examine the importance of the choice of the numerical value of $m_h$ the characteristics belonging to somewhat different values of $m_h$ are here represented in a few cases. Knowing the exact value of the "heart" mass appears not to be very important. Therefore in all cases except those of Figs. 3 and 5 $m_h$ is taken 1.5 Kg. only.

*4.* From the above it follows that it is understandable why the height of the peaks in Figs. 3 and 5 is so great for the given binding. The resonance peak at about 8 c/s means that occurrences appearing with this frequency will be represented more conspicuously than those closer to the heart frequency. However, in the experimentally determined BCG this frequency is hardly to be found. Because of this reason we suppose that the damping found by Grandell[3] from his experiments might be smaller than it is in the intact human body. Perhaps this is caused by the removal of the 2 costal cartilages.

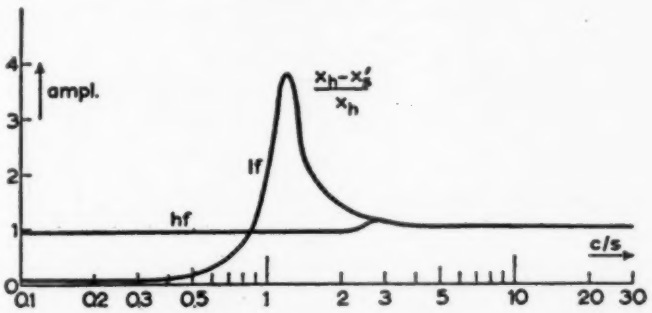


**Fig. 9—The ratio of the amplitude of the difference in displacement of "heart" and "subject" $|x_h - x_s'|$ and the amplitude of the displacement of the "heart" $|x_h|$, when a *low-frequency* bcg is used (curve *lf*) and when a *high-frequency* bcg is used (curve *hf*).**

From our calculations it followed that the height of the peak is very strongly dependent on the value of $\delta_h$, the ratio between the damping coefficient $\beta_h$ as it presents itself in reality and its critical value. From Grandell's paper[3] we deduced $\delta_h$ to equal 0.17. We erroneously made calculations also with $\delta_h = 0.11$, and the height of the resonance peak for $\nu = 8$ c/s appeared to be a few times greater than in the cases $\delta_h = 0.17$, represented in Figs. 3 and 5.

As we supposed the damping determined by Grandell to be too small, we calculated in one case the amplitude characteristic for a larger damping of the "heart," namely $\delta_h = 0.29$ (Fig. 14). We believe that it would be a good plan to repeat the measurements concerning the natural frequency and the damping of the "heart" with respect to the "subject." These should then be done without the removal of parts of the thoracic cage. Perhaps people with a splinter in the heart wall would be very good subjects.

*5.* As has been mentioned above, von Wittern[4] found for the natural frequency of the heart with respect to the thoracic cage 5 c/s. He determined this value in the following manner: From roentgenograms it appeared that the heart

in the upright position had shifted 1 cm. with respect to the ribs in the direction of the feet, as compared with the reclining position. However, in comparing these two cases the displacement is originated by the action of gravity on all organs in the trunk. Von Wittern may have found a larger displacement of the

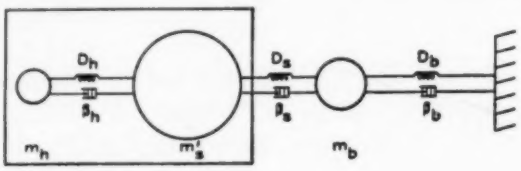


Fig. 10.—The system given in Fig. 1, *A* and *B*. The binding between the masses inside the outlined area is assumed to be infinitely strong in this case.

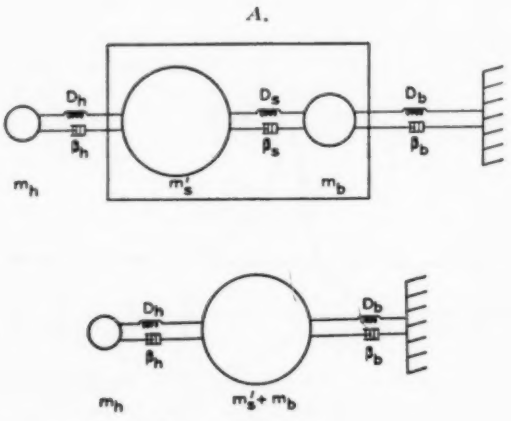


Fig. 11.—*A*, The system given in Fig. 1, *A* and *B*. The binding between the masses inside the outlined area is supposed to be infinitely strong in this case. *B*, A simpler illustration of the system given in *A*.

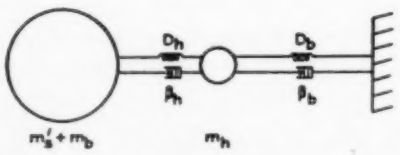


Fig. 12.—The same system as given in Fig. 11, *B*, except that the masses are interchanged.

heart than would be caused by a force working on the heart alone. Starting from this hypothesis and using von Wittern's formula[4] it is clear that he found a low natural frequency (5 c/s). For this reason we did not make use of the number given by von Wittern.

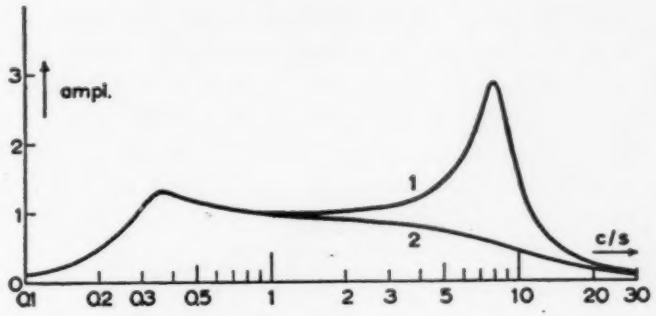


Fig. 13.—The amplitude characteristics of the systems represented in Figs. 11 and 12, if the displacement of the center of gravity $x_c$ is found by recording the displacement of the mass to the right in Figs. 11, *B* and 12 (curves 1 and 2, respectively).

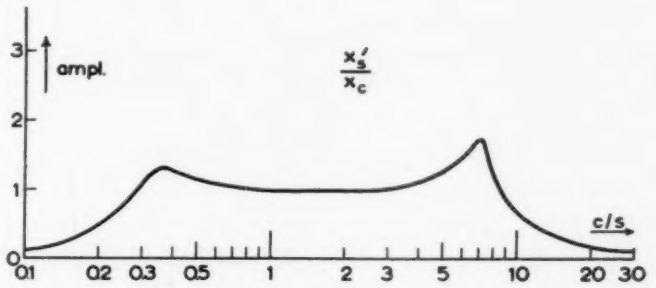


Fig. 14.—The amplitude characteristic of a *low-frequency* bcg if the displacement of the center of gravity $x_c$ is found by recording the displacement of the "subject" $x_s'$. The mass of the "heart" is chosen 1.5 Kg. The difference between this character stic and those represented in F g. 3 is only that the damping of the "heart" is chosen somewhat larger in Fig. 14.

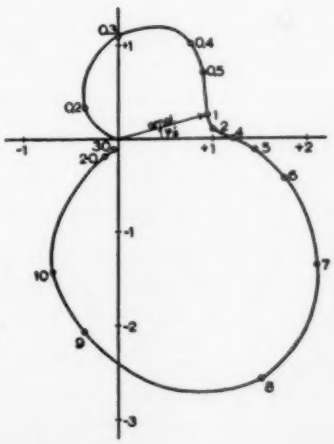


Fig. 15.—The amplitude and phase characteristics taken from Figs. 3 and 4 for the case that the mass of the "heart" equals 1.5 Kg. represented in one figure. The numbers along the curve represent the frequencies in c/s.

*6.* The representation of the displacement of the center of gravity within the subject, if it is recorded by a low-frequency bcg, is certainly better than is given by the characteristics of Figs. 3, 5, or 14. Not all displacements occur within a part of the body that is elastically bound to the skeleton. For instance, the arteries in the extremities may be assumed to be bound rigidly to the skeleton with respect to the binding between "heart" and skeleton. So the representation of the BCG will be better than is actually represented in the above-mentioned characteristics. This problem and the quantitative prediction of the BCG will be treated in a later publication.[7]

## CONCLUSION

Records of the ballistocardiogram are distorted by the weak binding between the heart with its surrounding tissues and the skeleton, and by the binding between the skeleton and the ballistocardiograph.

From the numerical treatment given in the present paper it follows that only the components of higher frequencies are distorted if a ballistocardiograph of the low-frequency type, e.g., according to Talbot[1] or according to ours,[2,5,6] is made use of (Figs. 3 and 5).

Moreover, it follows from this treatment that the ballistocardiogram is also distorted for frequencies closer to the heart frequency if a ballistocardiograph of the high-frequency type (according to Starr) is used. This is caused by the gliding of the subject over the ballistocardiograph (Figs. 7 and 8).

## SUMMARY

The influence of the binding between heart and skeleton and between skeleton and ballistocardiograph upon the representation of the human ballistocardiogram has been treated quantitatively for the low-frequency and the high-frequency ballistocardiograph.

It appeared that frequencies close to the frequency of the heart are more distorted when a high-frequency ballistocardiograph is used than when the low-frequency ballistocardiograph is the recording instrument. This is caused by the compliance between subject and ballistocardiograph as well as by that between the latter and the surroundings.

## APPENDIX

There are a number of methods for indicating the quality of representation of a phenomenon. To represent the distortion of the ballistocardiogram by the transducer systems we have chosen the method in which the amplitude and phase distortion are given separately.

As an example of another method of representation of the distortion, the results given in Figs. 3 and 4 will be represented in another way for the case $m_h = 1.5$ Kg. A common method in techniques called the Nyquist diagram will be applied for this purpose.

From the differential equations (1) to (3) it followed that

$$\frac{x_s}{x_c} = m_s\omega^2 \frac{s_1 + js_2}{n_1 + jn_2} = \frac{m_s\omega^2}{n_1^2 + n_2^2}(s_1n_2 + s_2n_1) + j\frac{m_s\omega^2}{n_1^2 + n_2^2}(s_2n_1 - s_1n_2). \qquad (15)$$

Formula (5) is to be found from this equation by calculation of the absolute values. Moreover, formula (10) follows from relation (15). Instead of evaluating the amplitude ratio $|x_s|/|x_c|$ and the phase shift $\varphi'_s$ apart, both can be found from one figure. On the rectangular axes of Fig. 15

the real part of the right side of formula (15) is plotted on the horizontal axis while the imaginary part is plotted on the vertical axis. For each frequency a point in the coordinate system is found. The frequencies in the range from 0 to 30 c/s give the curve represented in Fig. 15. For an arbitrarily chosen frequency the amplitude representation is found by measuring the distance from the origin of the coordinate system to the point of the curve belonging to that frequency. The phase shift for that frequency is found by measuring the angle between the just mentioned line of communication and the positive real axis. The angle is calculated positive in the direction of the arrow in Fig. 15.

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# THE INFLUENCE OF LEFT BUNDLE BRANCH BLOCK ON THE CLINICAL RESULT FOLLOWING AORTIC COMMISSUROTOMY

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LEFT bundle branch block is a well-established electrocardiographic entity. Early investigators[1,5] considered the abnormality a poor prognostic sign, although more recent reports do not share this view.[6,10]

Various reviews over the past two decades have analyzed the influence of left bundle branch block on the risks attending major surgery. Again, opinions vary as to whether it seriously alters the mortality and morbidity following general surgical procedures.[11-13]

The pertinent question now arises whether this condition alters the risk of or response to surgery on the heart itself. With this inquiry in mind the present communication summarizes experiences in 8 patients with left bundle branch block who underwent aortic commissurotomy for rheumatic aortic stenosis by the transaortic or transventricular routes.

## METHODS AND MATERIALS

The criteria for left bundle branch block were those of Wilson and coworkers.[14] The standard 12 electrocardiographic leads were used routinely.

Left heart catheterization was carried out in 4 patients by the method described by Goldberg and associates.[15]

The 8 patients were selected from a group of 178 with aortic stenosis operated on during the period extending from April 1, 1952 to June 31, 1956. The surgical techniques employed have been described previously.[16]

## CLINICAL PICTURE

There were 7 men and 1 woman, whose ages ranged from 29 to 59 years, with a mean of 46 years. Four of the patients were younger than 50 years of age. Six offered a history of rheumatic fever or its stigmata.

The symptomatic picture was characterized chiefly by the early development of fatigue and dyspnea. Paroxysmal nocturnal dyspnea appeared later. Angina was present in 3 patients, and syncope and ankle edema were noted in 2 cases. Pulmonary edema, vertigo, cough, and hemoptysis were not observed. Each patient described a history consistent with heart failure in the past.

The characteristic rough murmur of aortic stenosis was heard in the second right intercostal space in all patients, with typical radiation to the neck and left parasternal area. Three had an

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TABLE I

| NAME | RHYTHM | RATE | AXIS | ELECTRICAL POSITION | PREMATURE BEATS | P-R INTERVAL | QRS COMPLEX | | | | | RS-T SEGMENT | T WAVE | | Q-T INTERVAL |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | DURATION | | | SHAPE | | | | | |
| | | | | | | | QRS COMPLEX | VENTRICULAR ACTIVATION TIME | | R-R′ OR SLURRED R $V_5$-$V_6$ | TRANS. ZONE | $V_5$-$V_6$ | $V_1$-$V_2$ | $V_5$-$V_6$ | |
| | | | | | | | | $V_1$-$V_2$ | $V_5$-$V_6$ | | | | | | |
| A.W.B. | NSR | 94 | LAD | SH | — | 0.15 | 0.14 | 0.015 | 0.09 | Sl. RW | $V_4$ | Iso. | N | I | (0.348) 0.40 |
| J.H.S. | NSR | 84 | LAD | SH | FVPB | 0.20 | 0.15 | 0.01 | 0.12 | Sl. RW | $V_4$-$V_5$ | −1.5 | N | I | (0.366) 0.48 |
| G.L. | NSR | 86 | LAD | SH | — | 0.20 | 0.17 | — | 0.09 | Sl. RW | $V_4$-$V_5$ | −3 | N | I | (0.363) 0.37 |
| A.W. | NSR | 67 | LAD | HT | FVPB | 0.24 | 0.12 | 0.01 | 0.07 | Sl. RW | $V_5$ | −3.5 | N | I | (0.402) 0.44 |
| S.H. | NSR | 70 | LAD | HT | — | 0.20 | 0.145 | 0.015 | 0.11 | Sl. RW | $V_3$ | −1 | N | I | (0.395) 0.36 |
| S.E.B. | NSR | 72 | LAD | SH | — | 0.23 | 0.14 | 0.015 | 0.08 | Sl. RW | $V_4$-$V_5$ | −4 | N | I | (0.390) 0.41 |
| R.G. | NSR | 76 | LAD | SH | — | 0.17 | 0.135 | — | 0.10 | RR′ | $V_4$ | −1.5 | N | I | (0.392) 0.40 |
| T.A. | NSR | 75 | LAD | SH | FAPB | 0.24 | 0.15 | — | 0.09 | Sl. RW | $V_4$-$V_5$ | −1 | N | I | (0.374) 0.44 |

All the durations and intervals are expressed in seconds. The values reported represent the longest duration or interval. For the P-R interval, bipolar limb leads are considered; for the QRS complex duration, bipolar and unipolar limb leads are considered.

LAD = left axis deviation; HT = horizontal; SH = semihorizontal; FVPB = frequent ventricular premature beats; FAPB = frequent atrial premature beats; Sl. RW = slurred R wave; R-R′ = R-R′ wave; Iso. = isoelectric.

The sign (−) preceding the voltage indicates negativity. N = normal; I = inverted. The numbers in parentheses represent the upper limit of the normal for that rate.

early blowing diastolic bruit in the same region. The aortic second sound was diminished in 5 and absent in 3 patients.

The electrocardiographic findings are reported in Table I. Normal sinus rhythm was present in all cases. The apical rate ranged from 67 to 94 beats per minute, with an average of 78 beats per minute. All tracings demonstrated left axis deviation. The electrical position of the heart was semihorizontal in 6 patients, and horizontal in 2. The P-R interval ranged from 0.15 to 0.24 second, with an average of 0.203 second. In 3 patients, it measured greater than 0.20 second. The duration of the QRS complex ranged between 0.12 and 0.17 second, with an average of 0.144 second. The ventricular activation time in $V_1$ and $V_2$, where measurable, ranged between 0.01 and 0.015 second. In $V_5$ and $V_6$ it ranged between 0.07 and 0.12 second, with an average of 0.094 second. There was evidence in $V_5$ and $V_6$ of a slurred R wave in 7 cases, and a R-R′ in 1 case. Characteristic depressions of the S-T segments and inversion of the T waves were noted to the left of the precordium. The Q-T interval ranged between 0.36 and 0.48 second, with an average of 0.412 second for the average rate of 78 per minute. Only 1 patient had a Q-T interval within the limits of normal.

The cardiac contour was *en sabot* in all instances. One-plus enlargement of the left ventricle was present in 1 patient, 2-plus enlargement in 4, and 3-plus enlargement in 3 patients.

Left heart catheterization was carried out successfully in 4 patients, with evidence in all of a significant mean systolic pressure gradient across the aortic valve (Table II). In a fifth patient, the cardiac catheterization was unsuccessful because of paroxysmal atrial tachycardia. The other 3 cases were operated on before the era of left heart catheterization.

TABLE II. PRESSURE

| NAME | LA | LV | BA OR AORTA | GRADIENT |
|---|---|---|---|---|
| A.W. | (12) | 230/6 | 90/52 | 140 |
| S.H. | ( 9) | 180/16 | 140/80 | 40 |
| S.E.B. | (24) | 150/24 | 120/80 | 30 |
| R.G. | (10) | 186/16 | 140/80 | 46 |

Pressures and gradients are expressed in mm. Hg. The Left Atrial mean pressure, and the Left Ventricle-Brachial Artery or central Aorta mean systolic gradients are reported.

## RESULTS

*Hospital Course.*—Three patients were operated on by the transventricular approach, and 5 had a transaortic commissurotomy. An adequate opening was believed to have been obtained in all cases.

Two patients died of a cardiac arrest, one at the time of surgery, and the other on the eleventh postoperative day. A third patient died 12 hours after surgery as the result of a myocardial infarction.

*Follow-up Review.*—Two patients expired 12 and 32 months, respectively, after surgery. The first died because of cardiac failure during bacterial endocarditis, and the second expired suddenly without known cause.

Three patients were found to be in good health 6, 24, and 43 months, respectively, after cardiac surgery, with an average duration of life following commissurotomy of 24.3 months.

A statistical analysis of the operative mortality and follow-up results in patients with aortic stenosis without intraventricular conduction defects as compared to those with left bundle branch block is present in Table III.

TABLE III. FOLLOW-UP AND MORTALITY RESULTS

| DIAGNOSIS | NUMBER OF CASES | | OPERATIVE | | LATE | | TOTAL | |
|---|---|---|---|---|---|---|---|---|
| | TOTAL | FOLLOW-UPS | DEATHS | PER CENT | DEATHS | PER CENT | DEATHS | PER CENT |
| Aortic Stenosis | 170 | 134 | 36 | 21.2 | 26 | 19.4 | 62 | 36.4 |
| Aortic Stenosis and CLBBB | 8 | 5 | 3 | 37.5 | 2 | 40.0 | 5 | 62.5 |

## NECROPSY

Autopsy was performed on 4 of the 5 patients who died. The following data were obtained:

*A. W.*—The heart weighed 880 grams. There was marked hypertrophy of the left ventricle which was 20 mm. thick. The right ventricle was also hypertrophic and 10 mm. thick. The aortic valve was heavily calcified and presented evidence of surgical manipulation. It would easily admit one finger. The other valves were normal. The coronary arteries showed no signs of arteriosclerosis. Microscopically, the myocardium showed focal areas of myocardial atrophy. The lungs, both grossly and microscopically, showed evidence of massive atelectasis.

*S. H.*—(Abstract obtained from St. Luke's Hospital, Kansas City. Missouri.) The heart weighed 850 grams. The left ventricle was hypertrophic. The aortic valve was heavily calcified, and had an adequate opening. It showed evidence of endocarditis, with thick, hard, inflammatory exudate along the valve margins. The other valves were normal. The operative site of the aorta seemed to be involved, also, in an inflammatory process, and sutures in this area were lying loose in the base of a dissecting aneurysm which had broken through to the adventitious tissue. This seemed to be a process of several days' or weeks' duration.

*S. B.*—The heart weighed 790 grams. There were petechiae at the anterior surface of the left ventricle. This was hypertrophic and 21 mm. thick. The right ventricle was normal. The aortic valve was heavily calcified, with an opening of one and one-half fingers, and showed signs of surgical manipulation. The other valves were essentially normal. Cut sections of the myocardium showed recent hemorrhage involving the left ventricular wall and the anterior half of the septum. Microscopic examination of sections of the left ventricle and septum revealed recent hemorrhage, granulocytic infiltration, and loss of striation of the muscle fibers consistent with myocardial infarction. Careful inspection of both left and right coronary arteries was negative. The lungs showed patchy atelectasis, both grossly and microscopically.

*T. A.*—The heart weighed 700 grams. The left ventricle was hypertrophic, with a thickness of 20 mm. The right ventricle was normal. The aortic valvular pattern was obliterated by heavy calcification broken at two sites by surgical procedure. An adequate opening was present. The other valves were normal. The coronary arteries were normal. Microscopically, there was hypertrophy of the left ventricular myocardium. Areas of fibrosis with increased perivascular connective tissue were noted. Medium-sized blood vessels showed thickening of the media. Atelectasis of the base of the right lung and infarction of the right upper lobe were observed both grossly and microscopically.

## DISCUSSION

Initially, a localized or diffuse lesion involving the intraventricular conduction system was considered the only mechanism responsible for the production of bundle branch block. In 1939, Rasmussen[17] first stressed the role of an additional factor, hypertrophy and strain of the ventricle.

The pathogenetic factor in 8 cases reported may be that of hypertrophy and dilatation of the left ventricle. It is postulated that as the ventricular mass increased in size, scattered areas of atrophy and fibrosis developed which involved the normal pathways of depolarization. It should be recalled that as the myocardium hypertrophies, the diffusion distance to the center of the muscle fiber increases, leading to impaired oxygenation. Thus, ischemia in the region of the left branch of the bundle of His may be responsible for the retarded activation of the left ventricular wall.

A previous study has related the success of aortic commissurotomy in part to the type of valvular morphology encountered.[18] Although it has been noted that there is a grossly reciprocal relationship between the severity of the valvular disease and the ability to attain an adequate aortic orifice, subsequent clinical improvement frequently occurs despite marked calcification and thickening of the valve. In keeping with these observations, post-mortem studies in the patients with left bundle branch block did reveal that an improved opening was accomplished by commissurotomy, although complete restoration of valvular function was not attained.

It has been suggested by Messer[10] that the disparity in life span between the various etiologic classifications of heart disease with left bundle branch block is related to individual difference in cardiac work tolerance. Disease of the conducting tissue itself, therefore, was not considered the sole prognostic determinant. The early and late mortality in 170 patients with aortic stenosis, alone or combined with other insignificant lesions, was 36.4 per cent. The higher mortality in this small series with left bundle branch block suggests the importance of the conduction defect in determining the response of the patient to aortic commissurotomy. No equivalent correlation between the operative results and survival could be established on the basis of severity of symptoms, heart size, or electrocardiographic abnormalities other than bundle branch block.

Left bundle branch block does not absolutely contradict the attainment of a good clinical result. In fact, 3 of our cases were doing well many months after the performance of their aortic valve surgery. However, its presence should serve as an indication of increased immediate and later risk.

Possibly, in some cases, intraventricular block may represent a reversible phenomenon. This could conceivably follow an increase in oxygen delivery to the myocardium after relief of the obstruction at the aortic valve. To date, such a change rarely has been seen after other forms of heart surgery, and represents a most uncommon event.

## CONCLUSIONS

1. An evaluation of the results of aortic commissurotomy in 8 patients with rheumatic arotic stenosis and left bundle branch block is presented.

2. The early and late mortality for this group is 62 per cent as compared to 36 per cent in a larger series of patients with aortic stenosis without conduction defects subjected to commissurotomy.

3. Clinical, physiologic, and necropsy data are presented and a follow-up review detailed. Two patients expired 17 and 32 months, respectively, after surgery.

4. Left bundle branch block should serve as a relative contraindication to the performance of an aortic commissurotomy, although occasionally it is associated with a good clinical result.

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# DOUBLE OUTLET RIGHT VENTRICLE

## A Partial Transposition Complex

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COMPLETE transposition of the great vessels is not a rare lesion in autopsy series of congenital heart disease.[1] In recent years, clinical recognition has often been possible.[2] Partial transposition complexes are, however, much rarer and with the exception of the Taussig-Bing heart have excited little clinical interest.

The Taussig-Bing heart consists of complete transposition of the aorta and partial transposition of the pulmonary artery which overrides a defect of the membranous ventricular septum.[3] The purpose of this report is to describe 4 cases, representing 3 varieties of another rare partial transposition complex characterized by complete aortic transposition with the pulmonary artery in normal position. The description "double outlet right ventricle" has been applied to such anomalies since both great vessels arise completely from this chamber. The available information concerning similar cases has been compiled and the differential diagnosis considered. The anatomical types are morphologically variable although closely related embryologically.

Either aortic or pulmonary hypoplasia is almost always present, since transposition is probably due to unequal division of the truncus by the truncoconal septum.[4] Double outlet right ventricle, therefore, may be subdivided into: (a) hearts with true persistent truncus arteriosus arising wholly from the right ventricle; (b) the "Eisenmenger type" with aortic hypoplasia, coarctation, or atresia, with or without atresia of the mitral valve; and (c) the "Fallot type" with pulmonary hypoplasia, stenosis, or atresia.

The best known are those with a persistent truncus, since, in about one-fourth, the single vessel arises wholly from the right ventricle.[5] This entity will not be discussed. Case 1 is an example of the "Eisenmenger type." It differs from the "true" Eisenmenger since, in the latter, the aorta overrides the ventricular septal defect; in the present example the aorta arises wholly from the right ventricle and to the right of the pulmonary artery. Case 2 falls into the same category, but is complicated by absence of the aortic arch. The ascending aorta, supplying blood to the upper half of the body, is not connected to the descending aorta which is filled from the pulmonary artery via a patent ductus. Cases 3 and 4 are classified as "partial transpositions of the Fallot

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type" and differ from the tetralogy in that the aorta arises completely from the right ventricle.

## CASE REPORTS

CASE 1.—This 3-month-old Negro girl was admitted as an emergency case. History was unreliable. A murmur had been noted at 6 weeks of age. A questionable history of intermittent cyanosis was obtained. For several days prior to admission, respirations had been rapid and "pneumonia" was suspected.

*Physical Examination.*—Temperature was 99° F.; pulse, 160; respirations, 48; weight, 8 pounds, 11 ounces. The patient was undernourished, with rapid, grunting respirations. No cyanosis, clubbing, or respiratory obstruction was found. Cardiac dullness was increased to right and left. A systolic shock was felt along the left sternal border. Pulmonary second sound was moderately intense and single. A Grade 3 systolic murmur was localized at the second right intercostal space. Râles were heard at both lung bases and there was dullness at the right base posteriorly. The liver was 2 fingerbreadths below the costal margin. Weak radial pulses were felt but none were palpable in the lower extremities. Systolic pressures obtained by the flush method[6] were consistently 80 to 90 mm. Hg in both arms, but flushing of neither foot occurred until the cuff pressure had fallen to zero.

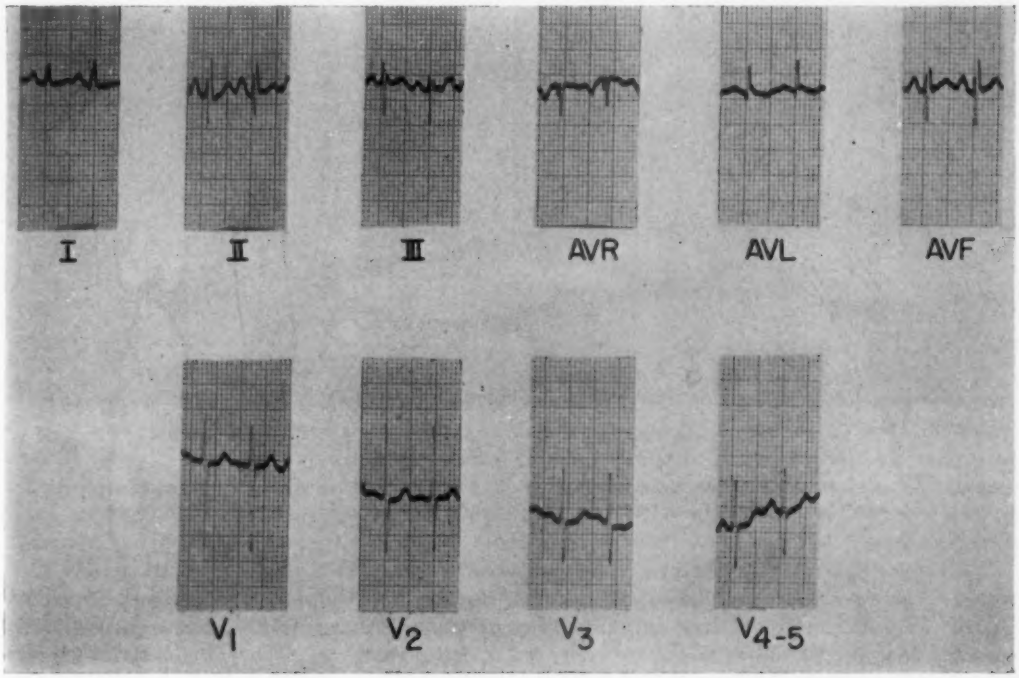
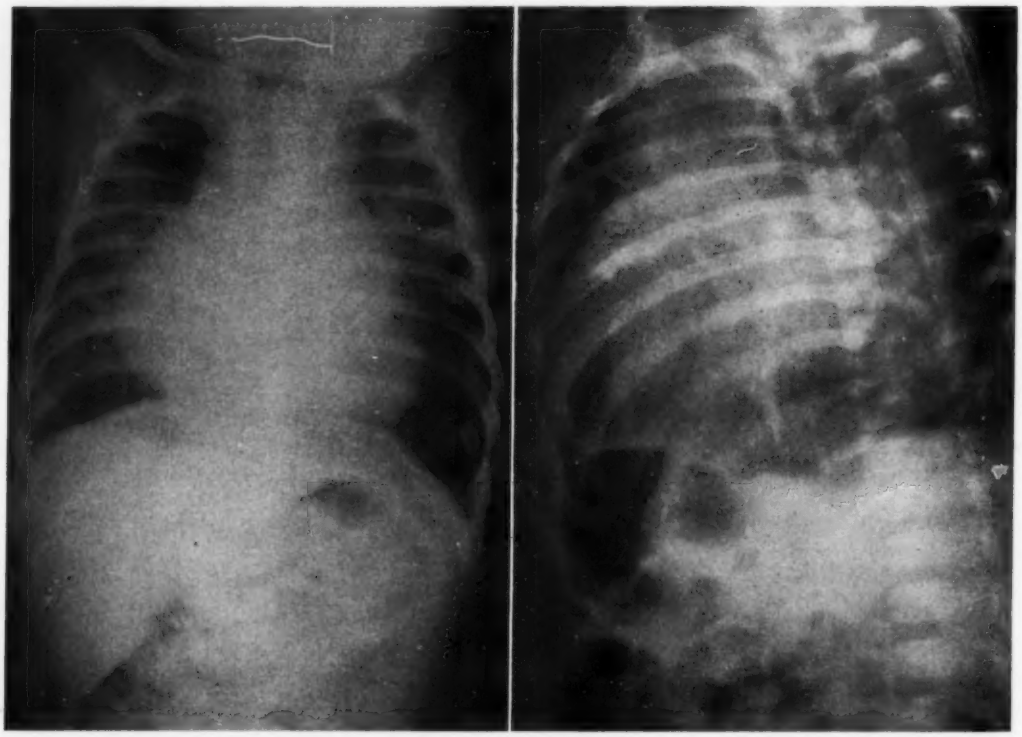



**Fig. 1.—ECG, Case 1. Interpretation in text.**

*Laboratory Data.*—Hemoglobin was 15.5 Gm.; erythrocytes, 4,120,000; electrocardiogram (Fig. 1) demonstrated a sinoauricular tachycardia at a rate of 150 per minute. The P-R interval was prolonged for age and rate (0.15 second). QRS duration was also somewhat prolonged for this age (0.08 second) and axis deviation was to the left. P waves were high and rather peaked. Deep S waves were present in precordial positions $V_1$ through $V_5$. The interpretation was combined hypertrophy, slight prolongation of auriculoventricular and interventricular conduction, and right auricular hypertrophy.

*Fluoroscopy.*—The heart was enlarged to right and left. The vascular pedicle was wide. Pulmonary "conus" was not definitely identified, but lung fields were congested and there were faint pulsations of the pulmonary secondary radicles. In the left anterior oblique, the vascular pedicle still appeared widened, the right ventricle was strikingly enlarged, and the left ventricle barely cleared the spine with the patient rotated 90 degrees. In the right oblique position, the "pulmonary conus" seemed full and pulsatile. X-rays (Fig. 2) were confirmatory.

The impression was that of severe aortic coarctation, septal defect, congestive failure, and probably pneumonia.



*A.* *B.*

**Fig. 2.—Anteroposterior and left anterior oblique films in Case 1. Note the rather wide supracardiac shadow in both views. Lung fields are plethoric and both ventricles appear to be enlarged.**

*Hospital Course.*—The child expired in pulmonary edema on the fifth hospital day.

The pertinent autopsy findings were limited to the chest. The lungs together weighed 120 grams, were noncrepitant, dark red, and their cut surface exuded fluid. The heart weighed 90 grams. Most of the anterior surface consisted of right ventricle. The pulmonary artery was greatly dilated but in normal position. The aorta, however, was small, and arose from the right ventricle to the right and slightly anterior to the pulmonary artery. Just distal to the origin of the left subclavian there was a sharp constriction of the aorta about 3 mm. in length. The smallest lumen had a diameter of 2 mm. A patent ductus arteriosus of 4 mm. internal diameter entered just distal to the coarctation. The descending aorta was smaller than usual. A 7 mm. defect of the membranous ventricular septum was found. The origin of the pulmonary artery was clearly to the right of this opening. Right and left ventricular walls were 5 mm. thick. The right ventricle was thought to be greatly dilated and hypertrophied, the left slightly. The left atrium appeared normal, but the right was moderately dilated and hypertrophied. A 5 mm. defect in the auricular septum near the foramen ovale was also found. All valves were normal. The coronary ostia were in their usual position. The most striking features are illustrated in Fig. 3. In summary,

these are: (1) complete transposition of the aorta; (2) coarctation of the aorta, "infantile" type; (3) patent ductus arteriosus; (4) atrial septal defect; (5) ventricular septal defect; (6) dilatation and hypertrophy of right auricle and both ventricles.

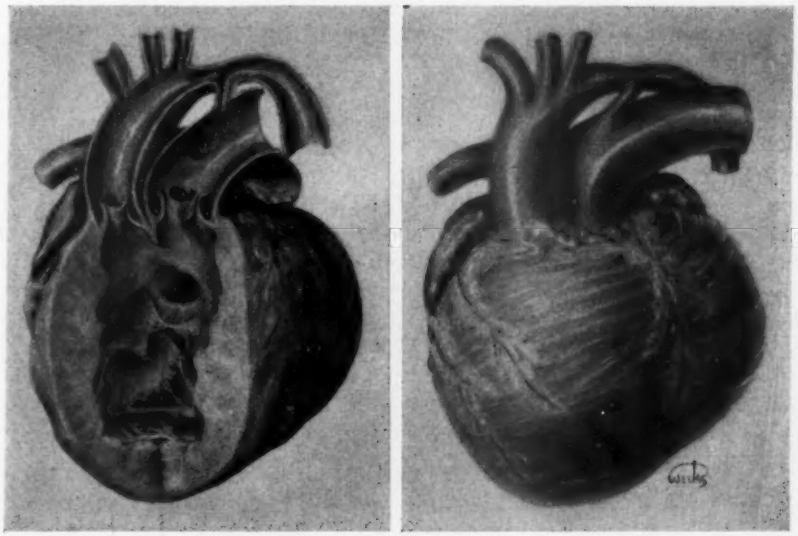


**Fig. 3.—Two drawings from the anterior aspect of the heart in Case 1. The aorta arises to the right of, and slightly anterior to, the dilated pulmonary artery. A small ductus arteriosus empties into the aorta distal to the coarctation. The three cusps of the normal tricuspid valve are seen below the ventricular septal defect. Both right and left ventricles were dilated and hypertrophied. The atrial septal defect is not visible in these drawings.**

CASE 2.—A 37-year-old white woman was admitted to Grady Hospital (Atlanta) with a ruptured uterus. A dead infant was found lying free in the abdominal cavity and was autopsied.

The body was that of a deformed white stillborn male, 34 cm. in length and weighing 3,420 grams. A Klippel-Feil deformity of the vertebrae and bilateral equinovarus were present. From the right ventricle arose a vessel directed straight to the neck; 1 cm. from its origin it divided into two vessels (right and left innominate) of equal size, each giving off a common carotid and subclavian artery. Coronary arteries arose normally from this vessel and followed their usual distribution. To the left of this vessel, which functioned as an ascending aorta, a large pulmonary artery also arose from the right ventricle. Small right and left branches came off this trunk as indicated in Fig. 4. The large patent ductus arteriosus was continuous with the descending aorta. No direct communication existed between ascending and descending aorta. The region of the ductus was not narrowed so that the pulmonary artery-ductus-descending aorta appeared as one vessel. The right ventricle was dilated and hypertrophied; its wall was 5 mm. thick. A high interventricular septal defect of 4 mm. was present. A muscular ridge partially divided the outflow tracts of the 2 great vessels. That of the aorta was small (3 mm.) and that of the pulmonary artery large (8 mm.). The left ventricle was a small, thick-walled chamber filled via the mitral valve; its only outlet was through the interventricular septal defect. The right atrium was dilated and about twice the size of the left, which was smaller than normal. The venea cavae entered the right atrium normally. The foramen ovale was patent to the probe. The left atrium received one large venous trunk from the right lung and two pulmonary veins from the left. Valve leaflets appeared normal.

The main features of this malformation are sketched in Fig. 4. In summary, these are: (1) small transposed ascending aorta; (2) absence of the aortic arch; (3) pulmonary artery continuous with the descending aorta via the ductus arteriosus; (4) ventricular septal defect; (5) single pulmonary vein from the right lung.

CASE 3.—A 27-year-old white housewife was admitted in labor. At delivery, the child's heart sounds were strong but spontaneous breathing never occurred. Heart action ceased after one hour.

Autopsy showed a well-developed newborn male, 47 cm. long, weighing 2.64 Kg. There was a 6 cm. cephalohematoma in the right temporoparietal area, but no external developmental anomalies. The lungs were airless, and diffuse petechiae were scattered over all surfaces. The liver was congested and weighed 105 grams.

The heart was slightly enlarged. The interatrial septum was completely absent, as was the upper third of the interventricular septum. There was a common A-V valve of 5 leaflets. The right ventricle was small, but thick-walled and the left was moderately dilated and thickened. The aorta arose from the right ventricle slightly anteriorly and to the right of the pulmonary artery. There was complete pulmonary atresia and no evidence of valve leaflets. The ductus arteriosus, however, was patent, dilated, and apparently filled the pulmonary artery fairly well since the size of this vessel and its branches was only slightly less than normal. In summary, the main features of the specimen are: (1) absence of the atrial septum (cor triloculare biventriculum); (2) common auriculoventricular valve (persistent atrioventricular canal); (3) ventricular septal defect; (4) transposition of the aorta; (5) atresia of the pulmonary orifice.

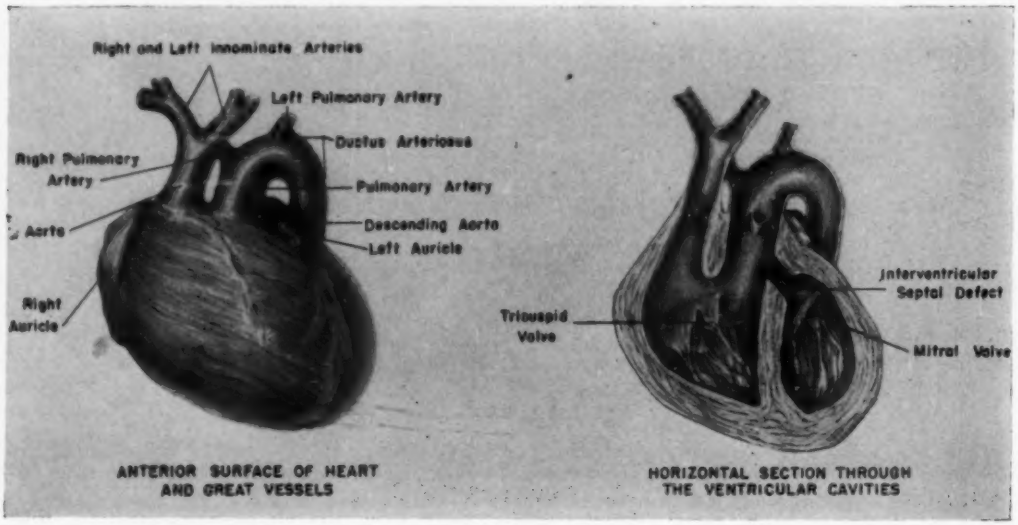



**Fig. 4.—Sketches from specimen in Case 2. Ascending aorta is strikingly transposed to the right and slightly anterior to the origin of the pulmonary artery. The muscle bundle separating the two outflow tracts is indicated. In this case, the direction of blood streaming from the ventricular septal defect appeared to be directly into the pulmonary artery. The pulmonary artery was continuous with the descending aorta via the ductus arteriosus.**

CASE 4.—This 3-month-old white girl was referred because of a murmur noted at 8 weeks of age.

*Physical Examination.*—Temperature was 100° F.; pulse 140; weight, 10 pounds; height, 22 inches. The patient was a poorly developed infant in no acute distress. Nail beds exhibited minimal cyanosis. A Grade 4 systolic murmur without a thrill was maximal at the third left intercostal spaces adjacent to the sternum. Pulmonary second sound was soft and single. Other findings were normal.

*Laboratory.*—Hemoglobin was 17 Gm.; electrocardiogram (Fig. 5) showed left axis deviation, accelerated conduction with P-R interval of 0.06 second and slurred initial portion of the QRS complexes (Wolf-Parkinson-White syndrome).

*Fluoroscopy.*—Estimated cardiothoracic ratio was 0.7. Lung fields were ischemic. In both obliques, the right ventricle appeared greatly enlarged. In the left anterior oblique view, the left ventricle also appeared displaced or enlarged posteriorly. X-ray is seen in Fig. 6.

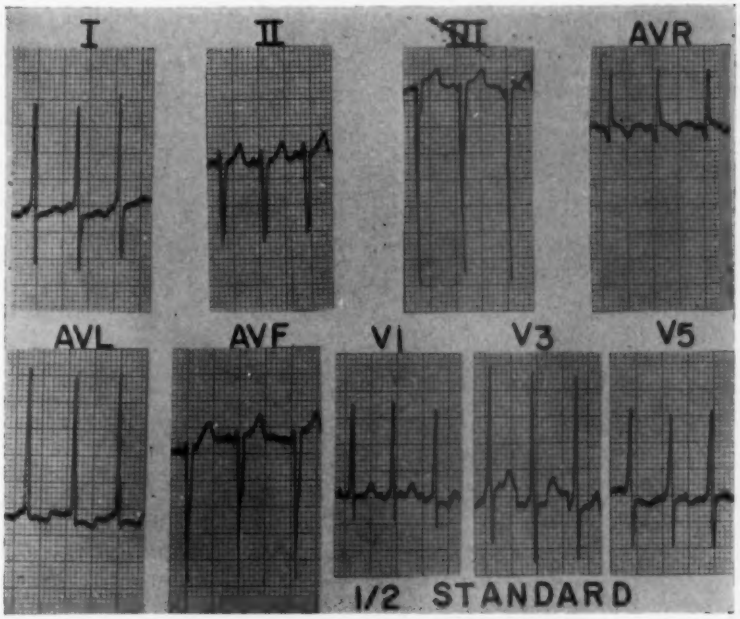



Fig. 5.—ECG, Case 4. Interpretation in text.

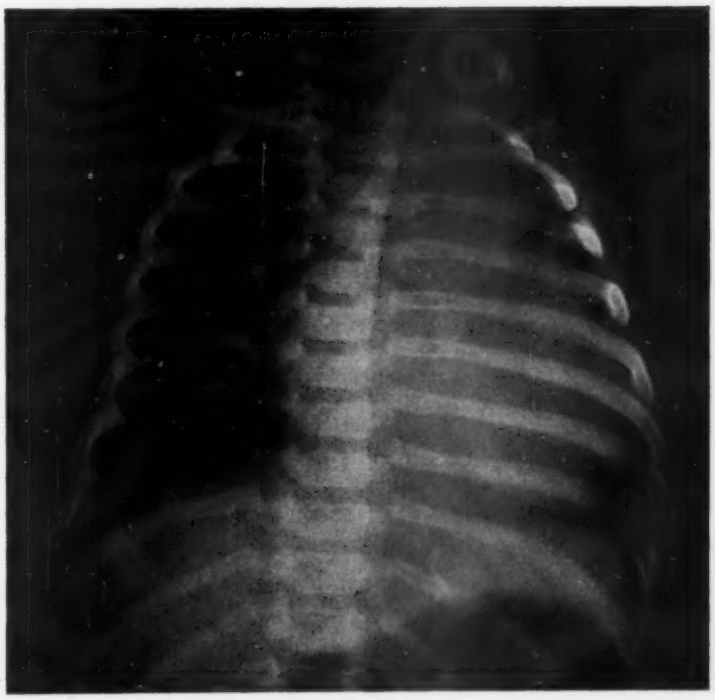


Fig. 6.—X-ray, Case 4, illustrating tremendous heart with poorly defined pulmonary arteries on the right.

*Course.*—The diagnoses considered were the tetralogy of Fallot, pulmonary stenosis with transposition, and tricuspid atresia. The mother was told to return the child in 1 month for further diagnostic studies. This was done, but the child had apparently been in congestive failure for 2 weeks and was dead upon arrival.

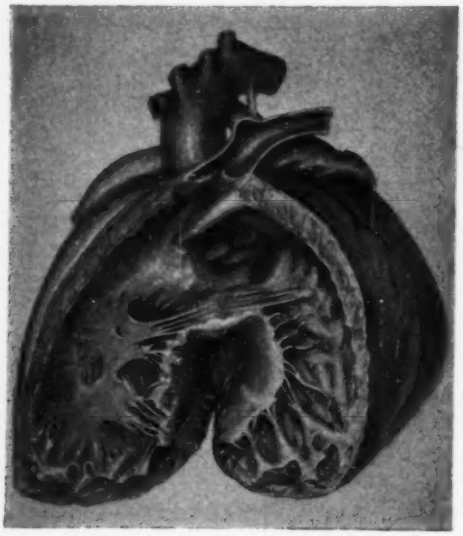


**Fig. 7.—Sketch of the specimen in Case 4. The aortic valve is seen to the right of the ventricular septal defect. Unlike Case 3, the aortic origin is definitely posterior to the pulmonary artery. Note the muscular ridge above the aortic valve, part of the crista supraventricularis, which "protects" the pulmonary outflow tract from blood streaming into the right ventricle from the septal defect.**

*Post-Mortem Examination.*—The heart weighed 100 grams and was grossly enlarged. From the anterior view, the aorta and pulmonary vessels seemed to be in normal position. The right ventricle was tremendously dilated and thickened, measuring 0.4 to 0.5 cm. The pulmonary artery was hypoplastic and the outflow tract obstructed by a large muscle mass partially forming the crista supraventricularis. A small infundibular chamber with a lumen of 2 mm. was present. The pulmonary valve was bicuspid, but did not in itself appear stenosed. An 0.8 cm. defect of the membranous ventricular septum was found. The pulmonary artery was in normal position. The aorta arose entirely from the right ventricle but, unlike Case 3, its root was posterior to the origin of the pulmonary artery. Aortic valve cusps were normal. The ductus was obliterated, and there was mild constriction of the aorta at the site of the ductus and slight dilatation proximal to this point. The left ventricular wall was dilated and hypertrophic, measuring 0.5 to 0.6 cm. The foramen ovale was patent to the probe. The lungs were congested. The liver was also greatly congested and weighed 162 grams. In summary, the main features, illustrated in Fig. 7 are: (1) ventricular septal defect; (2) infundibular pulmonic stenosis; (3) bicuspid pulmonary valve; (4) transposition of the aorta.

## DISCUSSION

One would expect the 3 varieties of double outlet right ventricle to present quite different clinical pictures. The anatomical features governing this would probably be the existence of pulmonary stenosis, patency of the ductus, and aortic coarctation.

Partial transposition, with and without pulmonary stenosis, has been recognized for many years and is included in the collections of Rokitansky,[7] Spitzer,[8]

Peacock,[9] and Abbott.[10] Little effort, however, has been directed toward clinicopathologic correlation. The "Eisenmenger type" has been described in detail only 3 times in recent years,[11-13] although interested pathologists have recognized others.[14] The recorded survivals indicate that this pathologic curiosity may also be a diagnostic problem.

The clinical details of the Eisenmenger type are summarized in Table I. Survival into the second decade may apparently be expected. Abbott[15] also noted 2 patients over 12 years of age.[15] As in Case 1, cyanosis may develop slowly. There may be no murmur, but a moderately loud parasternal systolic murmur is usually found. One example had an apical systolic murmur. Because of high pulmonary flow, a pulmonary insufficiency murmur is not unexpected.[11] $P_2$ is apparently accentuated. Electrocardiograms suggested right auricular and right ventricular hypertrophy in all. The left axis deviation of Case 1 seems unique. Prolongation of the QRS complex was present in two. Roentgenography shows plethoric lung fields and biventricular enlargement. The significance of the fairly wide supracardiac shadow in the anteroposterior view which remains wide in the left oblique is unknown because fluoroscopy reports are inadequate. The relative position of the aorta and the pulmonary artery (Fig. 3) may explain the finding. Slight rotation of the patient toward the right oblique should cause the shadows of the 2 vessels to become superimposed, resulting in a narrow vascular pedicle in this view. This sequence might differentiate the anomaly from complete transposition in which the superimposed great vessel shadows result in a narrow pedicle in posteroanterior view which widens in the oblique projections.[16] Coarctation and patent ductus are often present, but no "differential cyanosis" has yet been recorded. Ventricular septal defect will probably always be found. The peculiar hypertrophy of the crista supraventricularis has been almost universally described. This structure may serve as a septum dividing the outflow tracts of the pulmonary and systemic circuits as a result of which oxygenated blood from the septal defect streams out the aortic opening with relatively little mixing so that cyanosis is not striking. The mode of exodus was congestive failure in two and anoxia in another.

The differential diagnosis encompasses several conditions with cyanosis and plethoric lung fields. The most common is complete transposition which usually presents intense cyanosis from the neonatal period.[2] Almost all cases with the Taussig-Bing heart have been cyanotic at birth, also, but only one had evidence of left ventricular enlargement.[17] This anomaly may show a more prominent pulmonary artery in the anteroposterior fluoroscopic projection because of its levoposition. Cyanosis in the true Eisenmenger is usually later in appearance, with hilar dance striking, and the left ventricle normal.[18] The cyanosis of persistent truncus arteriosus may develop slowly and the condition must therefore be considered. Although the clinical picture is variable because of different anatomic types, continuous murmur, narrow vascular pedicle in all views, and the characteristic "shelving" of the right ventricle may be helpful.[5,19] Single ventricle without pulmonary stenosis also presents plethoric lung fields unless the pulmonary artery arises from a rudimentary ventricular chamber.[19]

TABLE I. DATA ON PARTIAL TRANSPOSITION COMPLEXES

| | FALLOT TYPE | | | | | EISENMENGER TYPE | | | |
|---|---|---|---|---|---|---|---|---|---|
| AUTHOR | BRAUN | GROSSE-BROCKHOFF | DOERR | CASE 3 | CASE 4 | SAPHIR | INGHAM | EDWARDS | CASE 1 |
| Age and Sex | 19 M | 12 F | 12 F | 1 hr. M | 3 mo. F | 21 M | 7 mo. M | 4½ mo. F | 4 mo. F |
| Cyanosis | Birth | First day, more in arms | Yes | Yes | Yes | "Blue baby" | 2 mo. with exercise | 2 mo. | 0 |
| Systolic Murmur | Second left ICS with thrill | Soft, second ICS and apex | ? | No | Grade 4, third left ICS; no thrill | Loud at apex and sternum | "Soft" over base | No | Grade 3, right second ICS |
| Diastolic Murmur | No | No | ? | No | No | Thrill and murmur, pulmonary area | No | No | No |
| $P_2$ | = $A_2$ | Loud | ? | ? | Soft, single | ? | ? | ? | Accentuated, single |
| Hemoglobin | 23 Gm. | 158 per cent | High (?) | ? | 17 Gm. | ? | ? | 13.2 Gm. | 15.5 Gm. |
| ECG | 1° Ht. Blk., I.V. Blk., RVH, RAH | I.V. Blk.; RVH, RAH | ? | ? | Wolf-Parkinson-White | ? | ? | RVH, RAH | LVH, RVH, RAH |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| X-ray or Fluoroscopy | RVH+, LVH+; Ischemic lungs | Slight increased size, RVH+ Ischemic lungs | Ischemic lungs (?) | ? | RVH, LVH; Ischemic lungs | ? | ? | Enlarged ht.; narrow vascular pedicle; plethoric lungs | Combined hypertrophy, pulmonary artery visible; plethoric lungs |
| Congestive Failure | Yes | No | ? | ? | Yes | Yes | No (?) | No | Yes |
| Associated Defects | Rt. aortic arch, ventricular septal defect, tricuspid and infundibular stenosis | Ventricular septal defect; infundibular stenosis | Two small ventricular septal defects; no further information | Common atrium; persistent A-V canal; vent. septal defect; pulm. atresia | Vent. septal defect; bicuspid pulm. valve; infundibular stenosis | Coarctation; patent ductus; vent. septal defect; large septal muscle bundle | Ventricular septal defect | Vent. septal defect closed by mitral valve leaflet; persistent L. Sup. vena cava; bicuspid pulm. valve; single coronary artery | Coarctation; patent ductus; vent. septal defect; aur. septal defect; septal muscle bundle |
| Remarks | Mongol | Postop. death | "Squatter" | No spontaneous breathing | Congestive failure | Died in failure | Died of pneumonia | Anoxic death | Died of pulm. edema |

**ICS = intercostal space. I.V. Blk. = intraventricular block. RVH, LVH = right and left ventricular hypertrophy. RAH = right auricular hypertrophy.**

Case 2 is similar to Case 1, but with an added defect. The fourth left aortic arch, which normally forms the transverse portion of the adult aorta has failed to develop. The ductus has persisted with undiminished size so that the pulmonary artery empties directly into the descending aorta.

Hamburger[20] reported a case with absent aortic arch and complete transposition in 1937. He found 13 cases of absent arch in the literature. One specimen, with aortic transposition, was similar to Case 2. Transposition was not described in the 10 subsequent cases recently summarized.[21] The majority survived only a few days. The exceptions are the cases of Gaspar[22] (6 months) and Dorney[23] (living at 5 years). Lev,[24] however, illustrates a specimen similar to Case 2 and has seen others. The full clinical picture in the rare case surviving infancy can only be hypothesized, but because of the position of the aorta the patient's upper extremities might be more cyanotic than the lower, as in Hamburger's case.

Partial transposition of the Fallot type (Cases 3 and 4) is also better known to pathologists than clinicians. It has been mentioned by several authors,[5,9,10,24] but most have probably masqueraded as the tetralogy of Fallot.[25] The only clinical descriptions found are summarized in Table I.[26-28] Expected longevity appears comparable to the tetralogy. Cyanosis appeared very early in all. Four had parasternal systolic murmurs in the pulmonary area (one thrill), one had an apical systolic murmur extending "bis in die diastole." The electrocardiograms suggested right auricular and ventricular enlargement and prolonged intraventricular conduction in all. Accelerated conduction (Wolff-Parkinson-White) has not been previously noted. Roentgenography demonstrated ischemic lung fields and prominence of both ventricles. Autopsy revealed ventricular septal defects, an infundibular stenosis or atresia, and a variety of other anomalies. This complex should easily be differentiated from the transposition complexes without pulmonary stenosis, but only with difficulty from the tetralogy or complete transposition with pulmonary stenosis. Case 4, however, demonstrated enlargement of the left ventricle which immediately suggested a transposition complex. Left ventricular enlargement in the tetralogy is, of course, very unusual.

It might reasonably be argued that Case 4 is embryologically no different from the usual tetralogy since the aorta is behind the pulmonary root (normal relationship) in contrast to Case 3 where the aorta arose anteriorly. Selzer[29] has pointed out that the spiral course of the interventricular septum causes the aorta to "override" an artificially created defect even in a normal heart. The great dilation of the right ventricular chamber may have displaced the septum to the left so that the aortic valve remained over only the right ventricle—i.e., an "acquired" rather than congenital aortic transposition.

Angiocardiography should demonstrate the extreme dextroposition of the aorta, and appears to be the best technique for identifying the transposition complexes. Proof of the feasibility of demonstrating the relationship of the pulmonary artery to the ventricular septum has recently been presented in a Taussig-Bing heart.[30]

SUMMARY

Three anatomic variations of a partial transposition complex characterized by pulmonary and systemic arterial circuits arising entirely from the right ventricle are described. Clinical data is presented in the first, the "Eisenmenger type," in which the heart revealed coarctation and complete transposition of the aorta, large pulmonary artery in normal position, ventricular septal defect, and patent ductus arteriosus. The clinical picture of this syndrome is characterized by slowly developing cyanosis, a parasternal systolic murmur, occasionally a diastolic murmur and signs of aortic coarctation. Survival may reach the second decade. Fluoroscopy reveals plethoric lung fields, combined ventricular enlargement, and a wide supracardiac shadow in the anteroposterior and left oblique views. Another example of this category was complicated by absence of the aortic arch. It was associated with a small separate ascending aorta, absence of the aortic arch, and descending aorta supplied by a patent ductus. All vessels arose from the right ventricle. Survival past the age of 6 years has not been recorded in this type.

The second principal variation of the "double outlet" right ventricle is the Fallot type. It resembles the tetralogy of Fallot because of various degrees of pulmonary hypoplasia. Biventricular enlargement, however, suggests that one is not dealing with a true tetralogy, and angiocardiography may demonstrate the exact relationship of the septal defect to aortic and pulmonary orifices.

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# Clinical Report

SUMMARY OF

## A FAMILIAL CASE OF WOLFF-PARKINSON-WHITE SYNDROME*

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A CASE of Wolff-Parkinson-White syndrome occurring in a medical student is reported. The case of a brother of this student who also had WPW syndrome had been reported previously by C. Erk.[1] Taking into consideration this anomaly as a congenital disorder, the remaining siblings have been examined. None of them showed any abnormal ECG.

The medical student who is the subject of this article has presented different kinds of ECG patterns on several occasions. The ECG has at times been normal, and on other occasions many ventricular extrasystoles or ventricular deflections with short P-R and prolonged QRS intervals appeared in the same strip. After exercise the patient has always developed paroxysmal auriculoventricular tachycardia which has been followed later by WPW. At different times the normal ventricular complexes have alternated with WPW complexes.

It is believed that in order to discover this congenital anomaly in suspected cases, several ECGs should be taken at different intervals, at rest and after exercise as well.

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*The original article appeared in Anadolu Klinigi, October, 1948. This summary is published here at the request of the author.

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# Special Committee Report

## SOCIAL CARDIOLOGY IN INTERNATIONAL PERSPECTIVE

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IN 1954, the International Society of Cardiology appointed a social committee as one of its instruments. Later, it was decided to include, also, problems concerning the rehabilitation of cardiac patients in the programme of this committee. The committee consisted of G. Biörck, Sweden, Chairman, H. K. Hellerstein, U.S.A., and S. Padmavati, India, vice-chairman, F. van Dooren, Belgium, Ch. Kossmann, U.S.A., B. Moia, Argentina, V. Puddu, Italy, W. Evans, United Kingdom, R. Froment, France, F. Grosse-Brockhoff, Germany, and J. Porto, Portugal. Valuable and stimulating contributions have also been provided by the following: A. Tybjerg Hansen, Denmark, P. Halonen, Finland, H. A. Snellen, the Netherlands, C. Müller, Norway, V. de Seijas, Spain, R. Garan, Turkey, J. D. Keith, Canada, P. D. White, A. Keys, and B. Williams, U.S.A., I. Chávez, Mexico, N. Kimura, Japan, A. H. Sorour, Egypt, F. Forman, Union of South Africa, and K. Maddox, Australia. The committee began its work during the Second World Congress of Cardiology in Washington, in September, 1954.

Its first task was to investigate and define those problems within the field of social cardiology that can be profitably dealt with by an international organization. For this reason, the committee has tried to collect information on the most important problems within the sphere of social cardiology encountered by the medical profession in all parts of the world. It is hoped that as the work progresses it will be possible to formulate recommendations of international validity. The ninth assembly of the World Health Organization recently adopted cardiovascular diseases as one of its major fields of interest. It is essential, therefore, that the opinion of the medical profession, and of the cardiologists in particular, should be well considered and clearly expressed. From the material obtained by the committee it is apparent, however, that the cardiologists' own understanding of the social aspects of "their" diseases is as yet limited. It may, therefore, be worth while to focus their attention on these problems, in the hope that further work will be stimulated for the years to come, until the next World Congress of Cardiology, in 1958.

**Paper read before the Second European Congress of Cardiology in Stockholm, Sept. 11, 1956.**
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## THE SCOPE OF SOCIAL CARDIOLOGY

While most of the programme of scientific meetings, generally, consists of studies of limited problems by refined techniques, and while most of our daily work is devoted to the treatment of the individual patients, there can be no doubt that another trend is slowly breaking through also in our specialty. With chronic diseases, such as those of the circulating system, the concept of a health-disease continuum is medically inescapable. In most countries, medicine no longer operates in a truly liberal society, where health is the concern only of the individual himself. It is quite obvious from our world-wide inquiry, that medicine is being increasingly fitted into the framework of society's planning for the citizens with regard to work, education, health, and recreation—in short, its welfare policy. In this regard, the concept of the health-disease continuum is equally inescapable. But society does not work only one way. More and more evidence is accumulating to show that culture and technique exert a considerable influence on the type and course of many diseases of the cardiovascular system. Thus, society may in its general settings aggravate biological conditions and, at the same time, by special measures try to ameliorate the harm already inflicted. If this concept is accepted, social cardiology will embrace not only the consequences of disease and invalidism, physically and psychologically, such as professional orientation, rehabilitation, and job replacement, workmen's compensation, and so on, but also those aspects of preventive medicine that apply to cardiovascular diseases, for disaster should no longer be passively awaited. Such attempts have been made with regard to rheumatic fever and rheumatic heart disease. They have aimed at the control of bacterial infection and have followed already established patterns in medicine. But the application of new epidemiological techniques will probably, in due course, raise the more formidable problem of the prevention of cardiovascular diseases by interference with fundamentals in our mode of living. When discussing the scope of social cardiology, one is forced to view the various technical matters against a general background of the interaction between the biology and the culture of man. There may be biological differences within the human races, which are important for the development of cardiovascular diseases. It is clear that differences in culture will place the emphasis within social cardiology differently. It will be seen, however, that these differences are not very great, and that they tend to diminish. This fact will increase the importance of rational international recommendations and activities in the field.

## THE PRESENT INVESTIGATION

In the beginning of the work of the committee, members were asked to give their opinions as to the most urgent problems in social cardiology and advice regarding a sensible approach to them. From the replies received it was obvious that technical recommendations had to be fitted into a general frame of knowledge concerning prevalence and trends in cardiovascular diseases as well as the medical organization in a number of representative countries. Questionnaires were prepared and distributed to committee-members and other interested persons. Altogether 21 answers were obtained from the following countries: *Europe:*

Belgium, Denmark, Finland, France, Germany, Italy, the Netherlands, Norway, Portugal, Spain, Sweden, Turkey, and the United Kingdom; *America:* Canada, U.S.A., and Mexico; *Asia:* India, and Japan; *Africa:* Egypt, and the Union of South Africa; and *Australia.* Several contributors supplied more information than was requested.

*Prevalence and Trend of Cardiovascular Diseases.*—All countries except 4 reported the availability of reasonably accurate data on cardiovascular mortality, while reliable data on cardiovascular morbidity was reported from only 3 countries, all of which recently performed large-scale health inventories. Figures of limited value, mostly derived from hospital populations, were reported from 13 countries, while 4 considered available data unreliable. In all countries cardiovascular diseases-at-large were considered one of the most important health problems, with a reported mortality from such diseases, including vascular diseases of the central nervous system, varying between 25 and 50 per cent of the over-all mortality. There were some differences of opinion regarding the trends in the incidence of cardiovascular diseases. A definite increase was reported by 8, while 3 would offer no opinion. The increase was ascribed to the increased incidence of coronary heart disease by 9, while 2 considered the age-adjusted rates rather unchanged. Eight countries considered rheumatic heart disease to have declined in frequency, while 1 considered it to be unchanged, and 1, to be increasing. Decline in syphilitic heart disease was reported by 6. It is apparent from this survey that diseases related to bacterial agents are more successfully dealt with than is arteriosclerotic heart disease. Whether or not the latter diseases are increasing more than can be accounted for by age-adjusted rates is subject to discussion, but the numerical increase is undisputed and its importance is clearly realized by the fact that, chiefly because of reduction of the juvenile mortality, the mean expectation of life at birth has risen by 17 years in the course of the last 50 years, and by 9 years during the last 25 years, in those countries for which data are available. This means roughly that in 18 of the countries the mean expectation of life at birth has risen from 49 years at the beginning of this century to 57 years in the 1920's and to 66 years at present. When these figures are broken down for men and women, the women are generally found to survive their husbands by 5 years. There are data indicating that the span between male and female mortality is increasing. The outstanding exception is India, where the mean expectation of life during the last 25 years has risen from 24 years to 30, and where husbands survive their wives by 5 years. Also in Egypt, life expectation is in the lower range.

*Health Systems and Their Provision for Social Cardiology.*—In all modern societies the responsibility for the care of acutely ill persons is assumed by the society in case the patient himself cannot afford the expenses. With regard to chronic illness, rehabilitation, and preventive services the situation is different. However, it is particularly in the latter fields that social cardiology has its applications. Consequently, it became necessary for us to investigate the extent to which the various systems of medical care did include such services.

In all countries except the United Kingdom the provision of medical care is divided between state, community, and private sources. All these countries,

with only two exceptions, report a tendency of growing state influence over the distribution of medical care. In most countries, wage earners, state employees, and indigents are covered by some kind of health insurance; only in 2 countries is the total population covered by such an insurance. On the preventive side, however, these systems chiefly cover mothers, children, and industrial workers, and it is overtly stated in several of the replies that cardiovascular diseases are not included in such services.

Certainly, legislation alone does not secure any medical care. It is even more essential to have a sufficient number of adequately trained medical personnel and of hospital resources. Six countries reported a sufficient number of hospital beds, and 8 an insufficient situation. Of the remainder, 2 considered the situation unsatisfactory for cardiac patients, 2 for the chronics and elderly sick, and 3 for rural or distant parts of the country. The supply of physicians was considered satisfactory in 9 countries, and not so in 2. For the rest, a shortage of cardiologists was reported in 5 countries, while in 4 others the distribution of practitioners was said not to respond to the real need. Shortage of nurses was reported also from several European countries. In all countries except 4, therefore, the possibilities for cardiac patients to receive adequate care were considered as unsatisfactory. The reasons for this opinion were classified as follows: economic in 10 instances, lack of personnel in 7, lack of equipment or beds in 5, lack of interest and education in patients in 3, and various other reasons in 3 instances.

Activities in the field of social cardiology, exemplified as rheumatic fever prevention, health education, and rehabilitation, were explicitly reported from 4 countries, while scattered or progressing activities were reported by 6 countries, and none by another 4. The main reasons for the slow development within this field was said to be mainly economical in 7 instances, and lack of interest on the part of both laymen and professional people in 9. In accordance with these observations 8 correspondents pointed to an intensified health education for both laymen and the medical profession as the most important way of improving the situation.

*Teaching and Research.*—It appears from the foregoing that the main obstacles to a complete cardiological service are threefold: weak national economy, lack of medical personnel, and lack of public and professional interest. In most instances, these factors are probably interconnected, and any shortage, either of money or of staff, will always be felt most strongly in the periphery. With the present concept of medical organization, prevention and aftercare are considered peripheral. However, national economics may improve more rapidly than does the supply of well-trained personnel, and for this reason several western countries today face an unexpected general shortage of professional personnel. The universal prolongation of life will, no doubt, present cardiologists with rapidly increasing problems, and unless the capacity of cardiological teaching and research centers is expanded, the deficiencies of today will be multiplied tomorrow. It is, therefore, necessary to look into the prospects within teaching and research in cardiology.

One of the serious problems delaying cardiological progress in some countries is a reluctance of the general internists who are often directors of departments of

medicine in universities and hospitals to recognize cardiology as an important specialty or even as a specialty at all. This was the prevailing situation in the U.S.A. a generation ago, but it has slowly yielded to natural evolution which has, however, not yet been universally accepted.

Out of the 21 countries from which answers to this questionnaire were received, 14 regarded the present graduate studies in cardiology as adequate, while 7 did not; 16 considered the possibilities for postgraduate training to be adequate, while 4 did not. Only 9 countries had sufficient refresher courses, while 10 had not. In one instance, facilities were considered adequate but not used by those who needed them most. Teaching in the social aspects of cardiology was considered to be inadequate in no less than 17 countries, chiefly because of a lack of interest and insufficient knowledge. With regard to research within the field of social cardiology, only 3 countries reported sufficient funds for such studies. (Incidently, these 3 countries are among those which also have the most advanced social legislation in the medical field.) The studies performed center around collection of statistical data, rheumatic fever prophylaxis, and the relationship of diet to arteriosclerosis. These studies are partly conducted by persons other than cardiologists.

Eighteen countries reported the need for more training of young doctors in the social aspects of cardiology; one correspondent believed the "consciousness" to be more wanting than the training, while another found the interest in his country not unsatisfactory and in the process of growing. As for the measures to improve the situation, there were a great many suggestions. They can be summarized briefly as the need for more research and fact findings, in collaboration with industry and public health administrators, in order to support improved and extended teaching on various levels, partly aided by an internationally organized training of personnel, including fellowship for junior workers and an exchange of visiting professors. There is also a great need for special training of nurses and social workers in this field.

*Problems of Work and Insurance.*—Early in the course of the committee's work, some problems presented themselves as amenable to immediate recommendations by an international body of cardiologists. Among these were workmen's compensation, life insurance regulations, and the employment of cardiacs, whether physically handicapped or not. All these problems are to a certain extent related to the difficulties in the determination of functional capacity and to the necessary change of traditional customs and points of view.

As regards workmen's compensation for cardiac disability, the connection between the work and the disease was regarded as difficult to evaluate by 3 correspondents, while difficulties to determine the physical capacity was stressed by another. Too short hospitalization time under the coverage of insurance was reported for 2 countries, while difficulties in finding jobs for the physically handicapped were mentioned in another 5. Only 7 of the countries had legislation which forced employers to use a certain percentage of the physically handicapped; in 3 of those countries cardiacs were not included, and in another, employers were said to be reluctant to employ cardiacs, preferring to pick persons in other categories. In 12 countries it was felt that more should be done along such lines—

only 2 correspondents considered the situation in their countries already satisfactory. Even more strongly was it felt, in 16 countries, that cardiacs in general were being unnecessarily turned down and restricted from employment, on meager evidence, and in several instances the state was considered to be worse in this respect than private business. Ten correspondents considered life insurance rejection and premium load principles too rigid, while 3 believed conditions to be adequate. Among the 10, 4 considered the premium load for elevated blood pressure to be unreasonably high.

Several suggestions have been forwarded regarding the improvement of the situation. A common denominator of some of them is the establishment of authoritative medical expert centers that could and should be consulted in matters concerning functional capacity and employment. If such bodies can be established on a scientific medical basis rather than on a basis of paper-evidence and bureaucracy, they might also influence professional and lay attitudes so that special legislation may be less necessary. It is only fair to state that research and practical measures with regard to rehabilitation of persons suffering from cardiovascular diseases at the present moment appear to be forceful and promising, particularly in the United States.

*General Comments.*—In this inquiry, it was repeatedly stressed that the size of the problems in social cardiology is largely unperceived by either the medical profession, the lay public, or the administration. There is a general lack of understanding of the mechanisms of chronic cardiovascular disease, and the consequences of the change in population structure and the panorama of diseases are not fully appreciated. To a considerable extent the medical development is hampered also by the conservatism of the medical hierarchy, which is slow to reorganize the branches of medicine for the new targets ahead.

Even those who realize the existence of a large working field in social cardiology find their activity restricted by the lack of sufficient knowledge, which is a consequence of the small volume of scientific studies on topics within this field. The lack of reliable morbidity statistics is a severe difficulty. Although it is generally stressed by the correspondents that there is too little teaching on social cardiology both within the profession and the lay public, this may at least in part be traced back to a lack of useful knowledge. To my mind, the time has come for an international conference of those who work in this field, in order to establish what is known and what is needed. In every branch of social medicine the application of scientific knowledge has to be built into the existing national systems of medical care and social legislation, but basic facts about cardiac function tests, the influence of the mode of life upon certain diseases, the techniques to prevent certain diseases, and experiences in the field of rehabilitation will largely be internationally valid.

There have also been a good deal of practical suggestions in the reports, such as compulsory employment of cardiac patients in sedentary jobs, building of health centers for old people, schemes for prophylactic treatment of rheumatic fever, organization of lay-professional heart associations (like the American Heart Association), financial drives for research money, and the publication of colorful booklets by the national societies. Some of these projects may meet with dif-

ficulties (or have already done so). In many of the countries, economic reasons have been indicated to explain why so little has been done. Those groups in a society which can hardly afford to pay for curative medicine are little likely to be able to pay for preventive medicine or, after their acute illness and consequent loss of earnings, for rehabilitation services, etc. In most countries, the medical profession works on a fee-for-service basis and is unwilling to accept salaried services except for work in public health or hospitals. For this reason, the "social" periphery of curative medicine attracts less interest. This is an issue that extends widely beyond social cardiology, and no solution shall be attempted here. Nevertheless, it is one of the main problems we have to face.

## CONCLUSIONS

After this review of what is thought and felt in 21 countries on the subject of social cardiology, some conclusions may be allowed.

On the basis of a program forwarded by Hellerstein, I would consider the following 10 points as the most important ones in social cardiology of today:

1. An international agreement on a uniform nomenclature and precise criteria for the diagnosis of cardiovascular disease, and expert supervision, through the national cardiological societies, of the current standards of diagnosis.
2. The establishment of the knowledge as to social and economic effects of heart diseases and disabilities on the individual and the society.
3. Research on the effect of environment, emotions, and occupations on the course of heart disease.
4. A program of prevention of heart disease (congenital, rheumatic, hypertensive, arteriosclerotic), and of prevention of the disabling effects of heart disease.
5. An international agreement on principles for standardized tests of cardiac function and physical performance.
6. Research on total energy demands of various occupations in different cultures, economies, and societies.
7. A scientific establishment of the principles of active rehabilitation from the acute illness, the convalescent period, and the return to the world of work.
8. Principles and technique of job modifications to fit the cardiac.
9. The establishment of criteria for the compensability of the cardiac.
10. The effects of forthcoming automation on the employment of the cardiac.

As is seen, research work is required in 8 of the 10 points, while in 2 instances (Nos. 1 and 5) international agreements on the basis of already existing knowledge might well be arrived at. Point 1 is at present subject to preparative work by the nomenclature committee of the International Society of Cardiology, under the chairmanship of V. Puddu. This work is hampered by a lack of funds. With regard to both these problems, a joint committee appointed by the World Health Organization and the International Society of Cardiology might, therefore, represent a fruitful approach. It is, likewise, apparent that international support of such national and joint programs as are suggested in the other 8 points should be of great value.

The social committee itself can only act by collecting material upon which it can form an opinion that can be expressed in the form of recommendations. It has been stressed by the correspondents that such recommendations, if presented to governments and other state authorities, to the heads and members of teaching units, and to the cardiological societies and the medical profession at large would have a definite value. Several resolutions were also proposed and decided upon by the social committee during its meeting in Stockholm in September, 1956. These resolutions have reference to the circulation of the committee's resolutions; avoidance of unwarranted cardiac invalidism arising from erroneous diagnosis connected with murmurs in school children, and cardiac pain and hypertension in adults; international standards for functional classification of cardiac patients; dissemination of current knowledge of the management of pregnancy in cardiac patients in all countries; conduction of a program of prevention of rheumatic fever by penicillin prophylaxis under the aegis of WHO; employment of patients with less severe forms of heart disease; special insurance and superannuation amenities for patients with heart disease; and voluntary national schemes for the care of cardiacs at home. The full text of these resolutions will soon be published.

In the program of the Third World Congress of Cardiology to be held in Brussels in 1958, a special session devoted to the social, economic, and industrial aspects of heart disease will be arranged. This will mean a definite opportunity of bringing social cardiology into the focus of interest of both physicians and laymen, an opportunity which should be seized upon by workers interested in this field.

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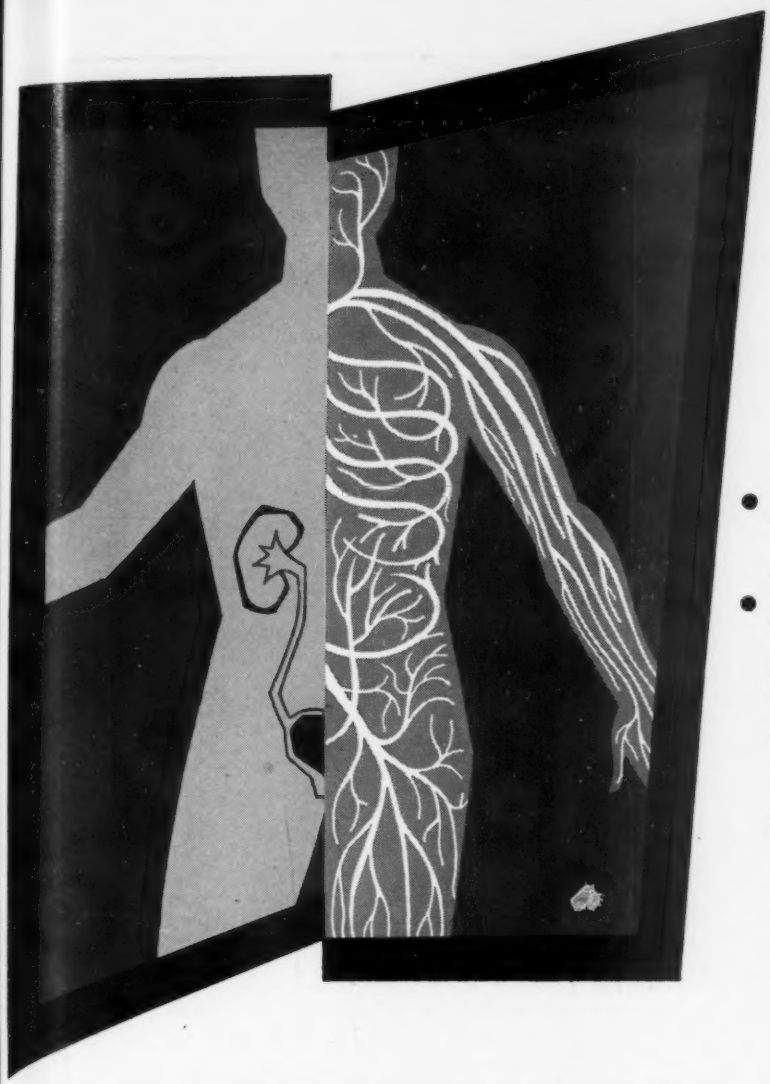

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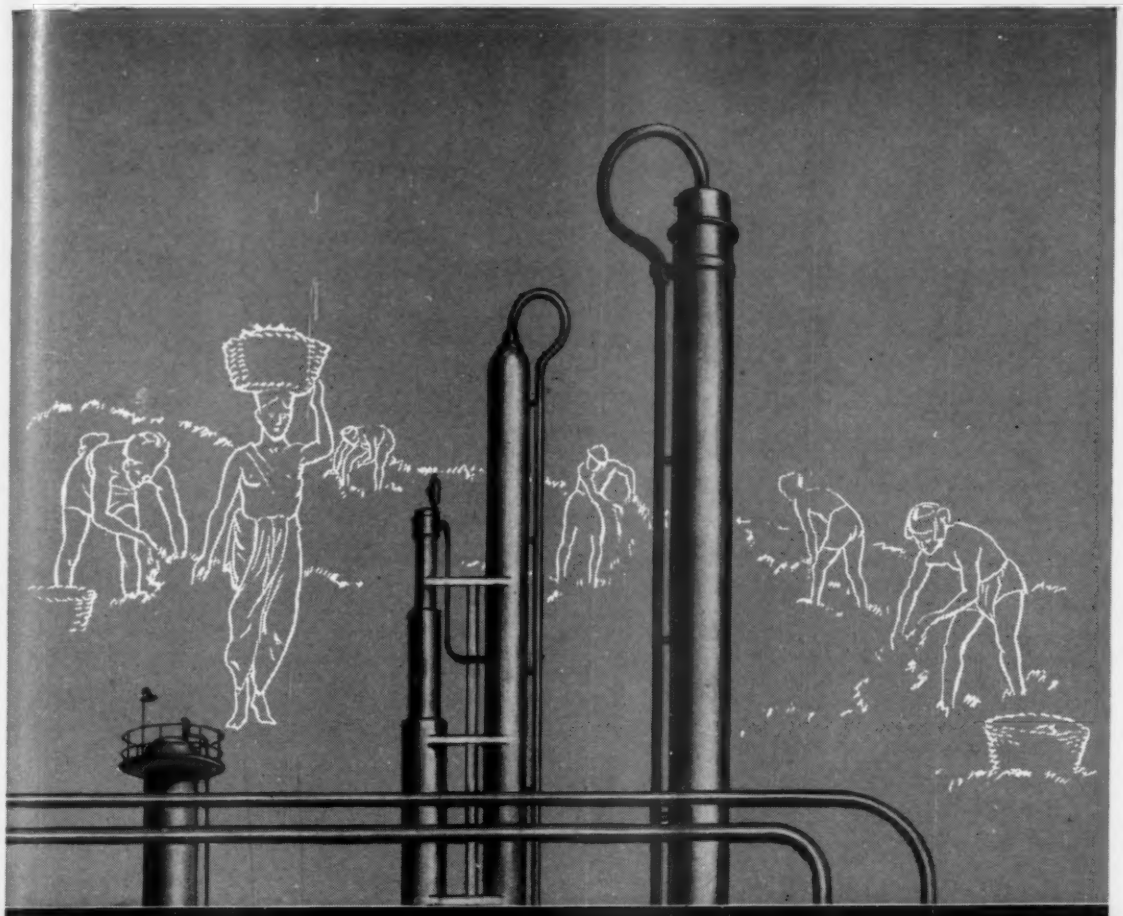

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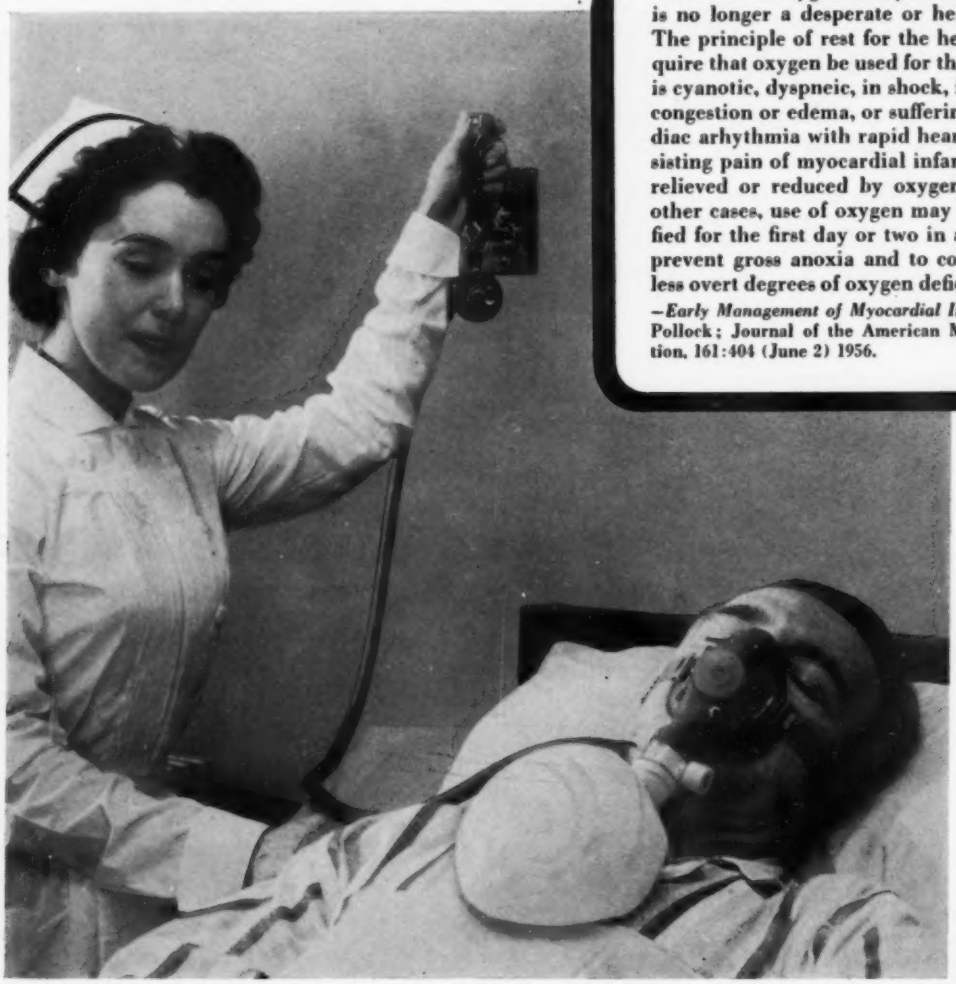

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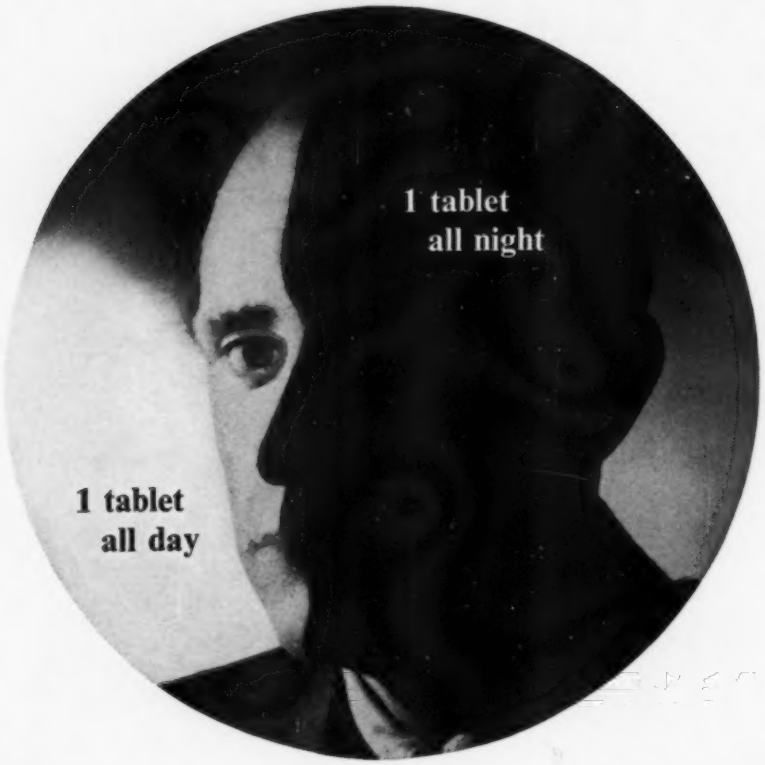


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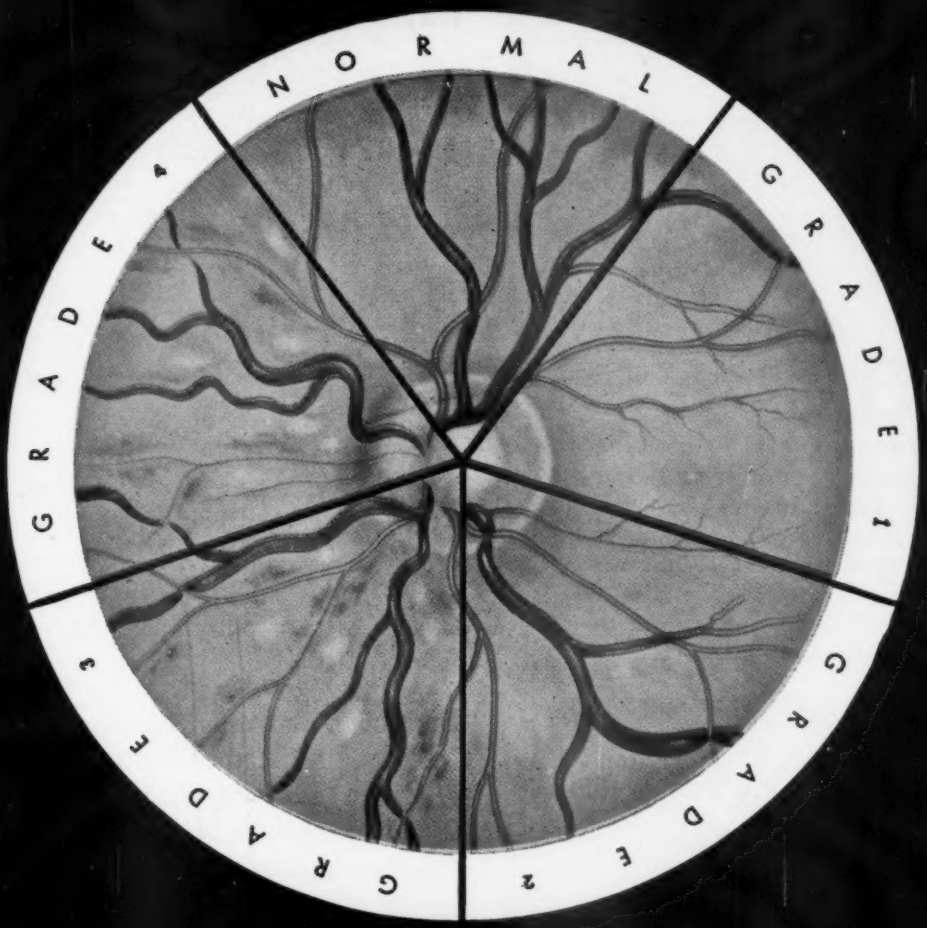


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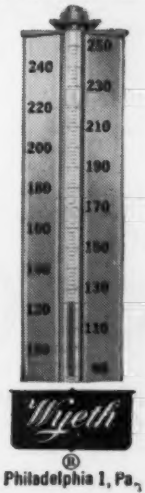


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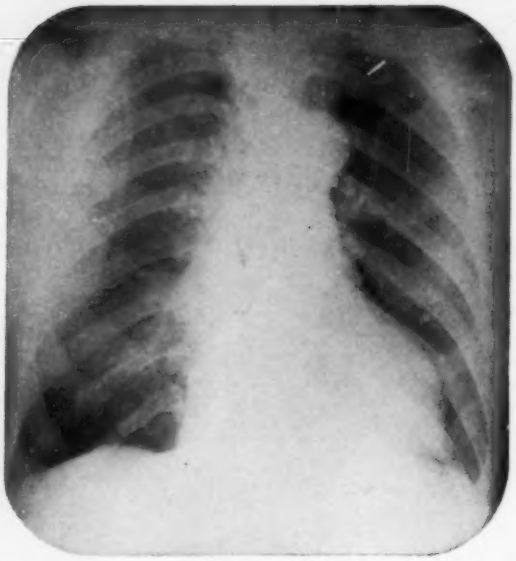

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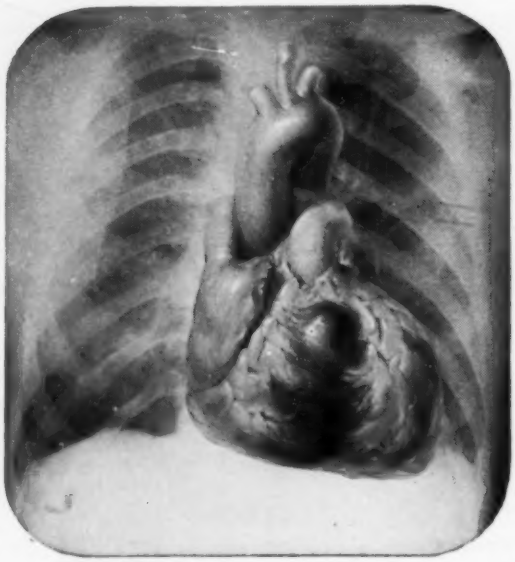

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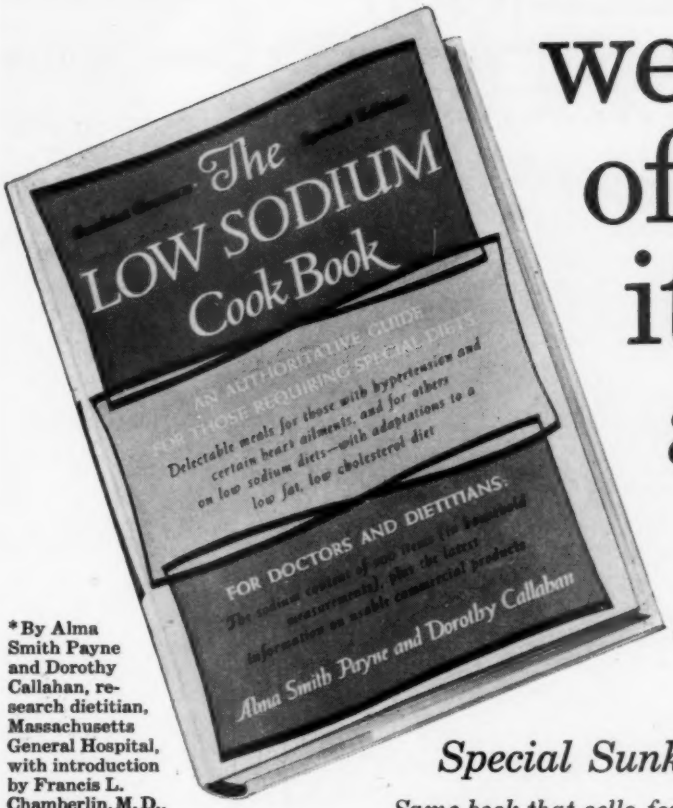
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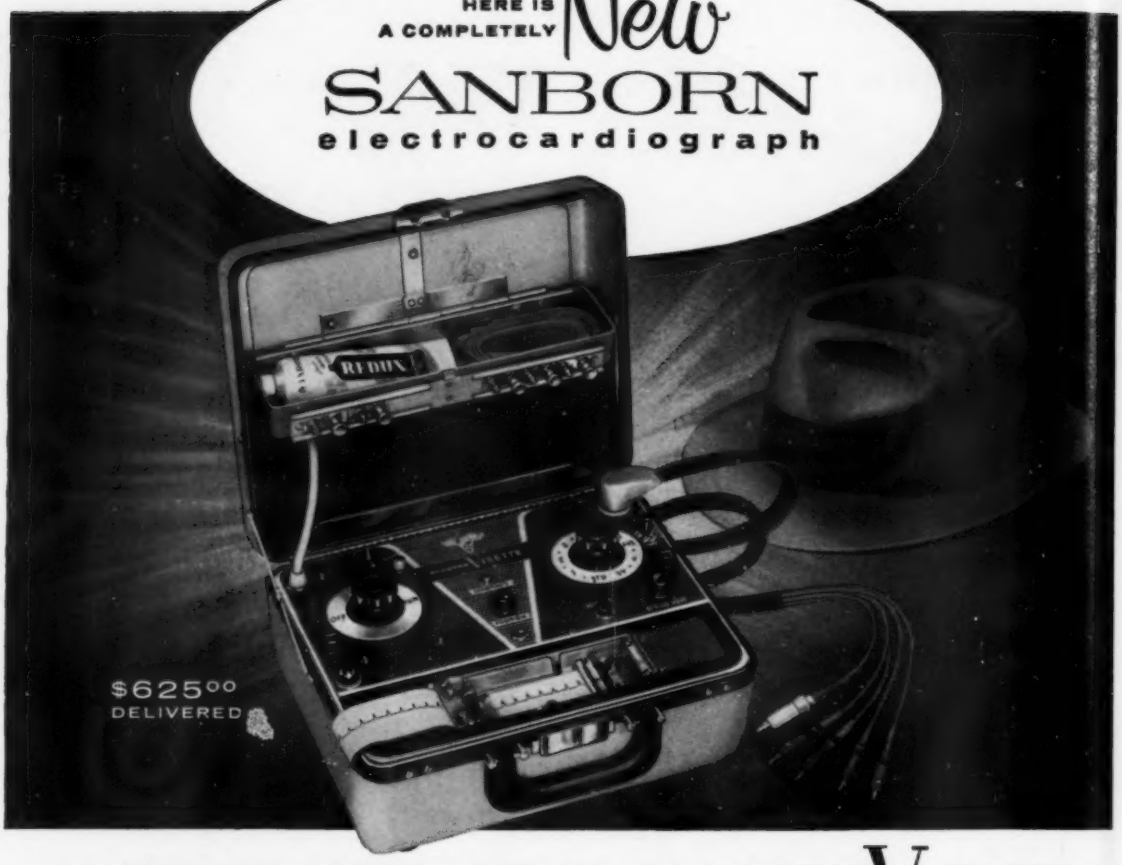


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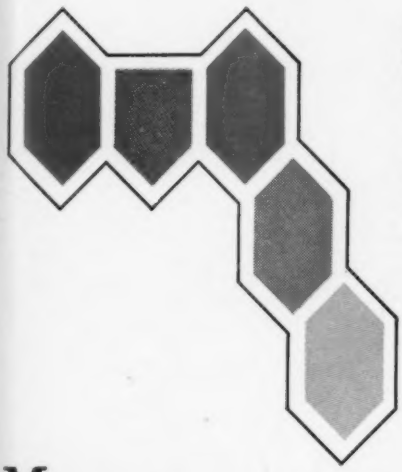

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Harmonyl proved particularly effective, for example, in tranquilizing a group of 40 chronically ill, agitated senile patients.[1]

Of particular interest is the observation that patients became more lucid and alert on Harmonyl therapy. And there was a complete absence of side effects with Harmonyl—although a similar group on reserpine developed such side effects as anorexia, headache, bizarre dreams, shakes, nausea and vomiting.

Following another eight-month study of chronic, hospitalized mental patients, Ferguson[2] stated:

- Harmonyl benefited at least 15% more overactive patients and proved more potent in controlling aggression—requiring only one-half to two-thirds the dosage of reserpine.
- Patients experiencing side reactions on reserpine often were completely relieved when changed to Harmonyl.

Ferguson concluded: "*The most notable impressions were the absence of side effects and relatively rapid onset of action with Harmonyl.*"

Comparative studies have shown Harmonyl and reserpine about equal in hypotensive effect. The tranquilizing action of the two drugs also appeared similar—except that few cases of giddiness, vertigo, sense of detached existence or disturbed sleep were seen with Harmonyl.

***Professional literature*** is available upon request. Harmonyl is supplied in 0.1-mg., 0.25-mg., and 1-mg. tablets. Abbott

References: 1. Communication to Abbott Laboratories, 1956. 2. Ferguson, J. T.: Comparison of Reserpine and Harmonyl in Psychiatric Patients: A Preliminary Report, Journal Lancet, 76:389, December, 1956. *Trademark

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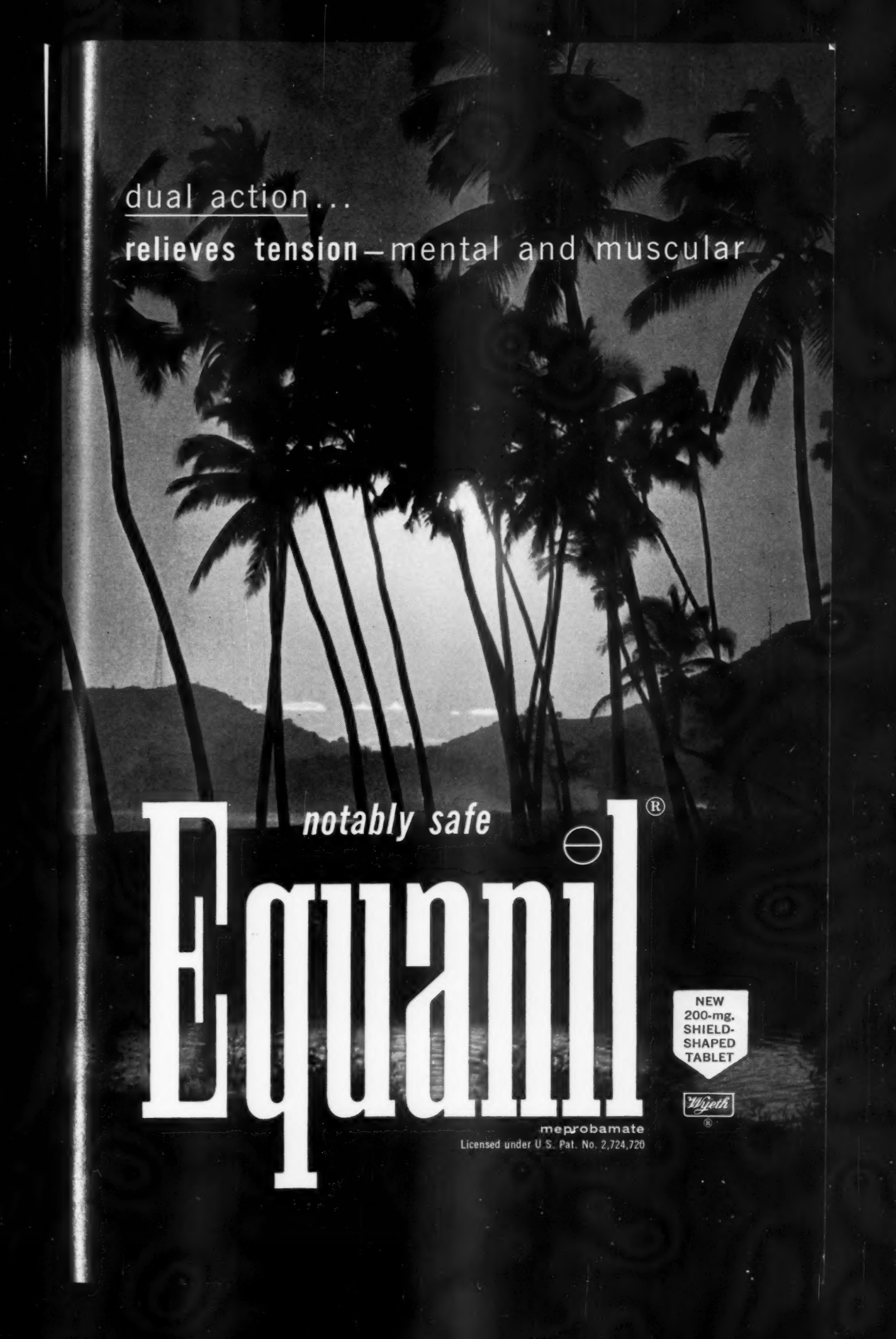
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# Serpasil

(reserpine CIBA)

*On the following pages you will find information on these aspects of Serpasil therapy:*

## The growing use of Serpasil in everyday practice


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| 3 | emotional disorders |
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| 4 | acute hypertensive crises |
| 5 | alcoholism |
| 5 | pediatric emotional problems |
| 5 | acute psychotic disturbances |
| 6 | side effects and precautions |

# in hypertension

## Serpasil® can always be considered first

**BECAUSE** *alone:* Serpasil successfully reduces blood pressure, slowly and safely, in about 70 per cent of cases of mild to moderate hypertension.[1]

**BECAUSE** *as a "primer":* Serpasil may be advantageously used to begin antihypertensive therapy, however severe the case, since it gently adjusts the patient to the physiologic setting of lower pressure.

**BECAUSE** *as a "background" agent throughout other therapy:* Serpasil permits lower dosage of the more potent antihypertensives needed for refractory cases, thus minimizing the incidence and severity of side effects.

USUAL DOSE: Initially, two 0.25-mg. tablets. After a week, daily dose should be reduced to 0.25 mg. or less for maintenance.

*"...a useful agent for the treatment of certain types of hypertension....The action...was increased by combining it with [Apresoline]..."*[2]

1. Coan, J. P., McAlpine, J. C., and Boone, J. A.: J. South Carolina M. A. 51:417 (Dec.) 1955.
2. Winsor, T.: Ann. New York Acad. Sc. 59:61 (April 30) 1954.

# in emotional disorders

## Serpasil® provides true emotional control

In your daily practice there are undoubtedly many patients whose degree and type of emotional disturbance—characterized by overexcitation, anxiety and agitation—can *not* be adequately controlled with sedatives or weaker tranquilizers. These are the patients whom you can help most with once-a-day administration of Serpasil. For Serpasil actually sets up a "stress barrier" against anxiety and tension the patient would otherwise find intolerable. With Serpasil you can control the emotional turmoil of disturbed individuals; and because Serpasil is restricted to prescription use, control remains in your hands.

***Although it is a first choice in hypertension, Serpasil does not significantly lower blood pressure in normotensive patients.***

USUAL DOSE: Initial range is 0.1 mg. to 0.5 mg. (two 0.25-mg. tablets) daily. As little as 0.1 mg. is sufficient for maintenance in some patients. Serpasil can be given in a single daily dose.

*"...relieves anxiety and irritability and calms the patient so effectively that because of this latter property alone, the drug should remain in the medicinal armamentarium."*

Finnerty, F. A., Jr., and Sites, J. G.: Am. J. M. Sc. 229:379 (April) 1955.

# in tachycardia

## Serpasil® slows the rapid heart

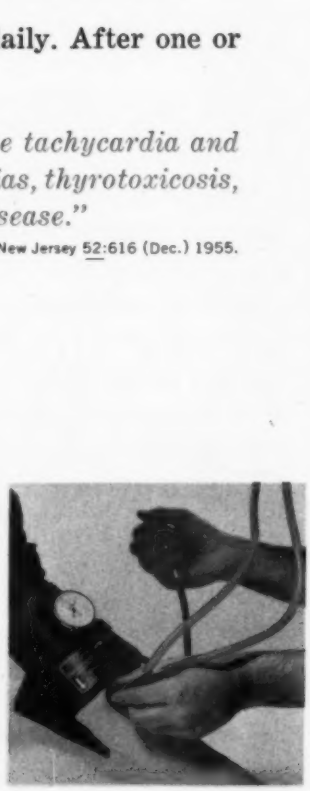
Many patients can benefit from the heart-slowing action of Serpasil. Those in whom tachycardia is deleterious are helped by its unique bradycardic effect, for Serpasil prolongs diastole and allows more time for the myocardium to rest. Blood flow and cardiac efficiency are thus enhanced.

USUAL DOSE: 0.1 mg. to 0.5 mg. (two 0.25-mg. tablets) daily. After one or two weeks dose may be reduced.

*"Reserpine [Serpasil] was found useful in relieving the tachycardia and emotional symptoms associated with cardiac arrhythmias, thyrotoxicosis, neurocirculatory asthenia, and even coronary heart disease."*

Halprin, H.: J. M. Soc. New Jersey 52:616 (Dec.) 1955.

# in acute hypertensive crises



## Parenteral Serpasil

Serpasil can be used alone in hypertensive emergencies or as a background to more potent antihypertensive agents. Its antihypertensive action is prompt and well-tolerated.

USUAL DOSE: 2.5 mg. (1 ml.) intramuscularly. Additional intramuscular doses of 2.5 mg. may be given as necessary every 8 to 24 hours.

*"...appears to be [a] treatment of choice for hypertensive crises."*



Griffin, R. W., Stover, J. W., and Ford, R. V.: New England J. Med. 254:593 (March 29) 1956.

# in alcoholism

## Serpasil® relieves drink-inducing tension

As a part of long-term therapy, oral Serpasil helps the alcoholic "stay on the wagon" by relieving drink-inducing tension, making him more amenable to your counseling.

In acute alcoholism, delirium tremens can generally be controlled within 24 hours with parenteral Serpasil...without the addicting or soporific dangers of drugs such as paraldehyde.

USUAL DOSE: *Chronic phase:* two 0.25-mg. tablets or less daily. *Acute phase:* two 2.5-mg. parenteral doses (1 ml. each) 3 or more hours apart. Occasionally, repeat injections of 2.5 mg. every 4 to 6 hours may be necessary.

*"...the tranquilizing and anxiety-relieving properties of this drug [Serpasil] offer the possibilities of its being extremely helpful for the long-term therapy of the chronic alcoholic."*

Greenfield, A. R.: Am. Pract. & Digest Treat. 7:241 (Feb.) 1956.

# in pediatric emotional problems

## Serpasil Elixir benefits the "problem child"

Serpasil provides a shield against stress in the overreactive, tense, "problem child." Striking remissions have been observed in children with excessive crying, poor eating and sleeping patterns.

USUAL DOSE: 0.1 to 0.3 mg. daily (½ to 1½ teaspoons of Serpasil Elixir, 0.2 mg. per 4-ml. teaspoon).

*"...provided dramatic relief in remitting the syndrome of irritability in 29 of the 32 cases studied..."*

Talbot, M. W., Jr.: Ann. New York Acad. Sc. 61:188 (April 15) 1955.

# in acute psychotic disturbances

## Parenteral Serpasil

The family physician is often called to subdue and arrange for quick hospitalization of patients who suddenly experience violent psychotic episodes. With intramuscular Serpasil these patients are quickly tranquilized and rendered amenable to 'quiet' hospitalization.

USUAL DOSE: 5 mg. intramuscularly followed, if necessary, by another 5-mg. intramuscular dose in 90 minutes.

*"It is now possible to discreetly manage acutely disturbed psychiatric patients by the prompt administration of adequate doses of reserpine (Serpasil)."*

Ayd, F. J., Jr.: The Pharmacologic Management of Everyday Psychiatric Problems (A Scientific Exhibit). Presented at the Clinical Meeting of the American Medical Association, Boston, Mass., Nov. 29-Dec. 2, 1955.

# Serpasil®: side effects and precautions

The side effects of Serpasil are characteristic of all rauwolfia preparations.

Although millions of patients have taken Serpasil over the past several years, very few serious side reactions have been reported. There have been no cases of blood dyscrasia, liver damage, addiction or withdrawal symptoms. When patients are properly selected and the lowest effective maintenance dose is established, the physician can prescribe Serpasil confidently, with little fear of untoward reactions.

## Depression

Mental depression, which has developed in a small percentage of patients treated with rauwolfia, should be differentiated from the transient change in mood or physical fatigue that is experienced by almost everyone in the general population. It should also be distinguished from the lethargy experienced by some patients on rauwolfia therapy.

In the few cases in which mental depression does occur, there is some question as to whether or not it is a direct effect of rauwolfia. According to Mayo Clinic investigators,[1] the evidence indicates that rauwolfia *per se* does not cause depression, but rather that it unmasks an underlying susceptibility to depressive reactions. Kinross-Wright[2] states: "It is likely that depression will occur only in a predisposed individual or in one who is already mildly depressed." Ayd,[3] in a very recent paper, states: "That this drug may cause depression is uncertain. After reviewing a large number of so-called drug-induced depressions it appears that in some cases what was called depression was excessive tranquilization, while in the rest, the patients were depressed before the drug was started, and what the drug did was make the depression more apparent."

Whether or not it is an effect of rauwolfia, physicians and responsible members of the patient's family should be on the alert for the development of symptoms of depression, particularly in patients with a history of pre-existing depressive tendencies. Daily doses above 0.25 mg. are contraindicated in the latter group. On withdrawal of rauwolfia, depression usually disappears, but active treatment, including hospitalization for shock therapy, has been required in some cases.

Adjunctive use of mood-elevating agents such as Ritalin is often sufficient to reverse mild depressions or drug-induced lethargy.

## Other side effects

In addition to lassitude or drowsiness, other mild side effects of Serpasil include occasional nasal stuffiness and increased frequency of defecation and/or looseness of stools. Rarely, anorexia, headache, bizarre dreams, nausea and dizziness occur. With parenteral Serpasil there is a possibility of marked hypotensive effect; therefore, the blood pressure should be taken before injection and the patient kept under observation for 5 or 6 hours thereafter. Because initial doses above 0.3 mg. tend to increase gastric secretion of hydrochloric acid, daily doses above 0.25 mg. are contraindicated in patients with a history of peptic ulcer and lower doses should be used with caution.

For further details on side effects and precautions, write Medical Service Division.

1. Litin, E. M., Faucett, F. L., and Achor, R. W. P.: Proc. Staff Meet., Mayo Clin. 31:233 (April 18) 1956.
2. Kinross-Wright, V.: Wisconsin M. J. 55:1073 (Oct.) 1956.
3. Ayd, F. J., Jr.: Presented at the Sesquicentennial Convention of The Medical Society of The State of New York, New York City, Feb. 18, 1957.

**SUPPLIED:**

**TABLETS**, 0.1 mg., 0.25 mg., 1 mg., 2 mg. and 4 mg.

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## A

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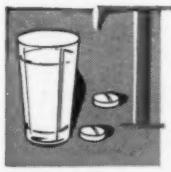

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Disorders of Rate and Rhythm.
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E.
Summary of QRS-T Patterns in Unipolar Thoracic and Limb Leads.

This work is the outgrowth of previous manuals prepared to supplement the Graduate Course in Electrocardiography given at Wayne University College of Medicine for many years. The objective is an organized presentation of the interpretation of the unipolar electrocardiogram. The only basic physics included is that considered essential to the interpretation of the tracing; controversial theories that do not contribute to clinical analysis have been omitted.

The analysis of the arrhythmias is made from leads that clearly display auricular as well as ventricular deflections; the interpretation of the QRS-T complex is based upon the findings in multiple precordial and back leads, supplemented, when necessary, by esophageal and unipolar limb leads. The registration of the normal and abnormal QRS-T complex is based upon concepts developed by Frank N. Wilson and associates and shown to be consistent with experimental and post-mortem findings. These concepts have been presented in detail and suitably illustrated with diagrams as an introduction to clinical analysis. Efforts have been concentrated on a systematic lucid description of electrocardiographic findings in an attempt to make reproduction of tracings unnecessary.

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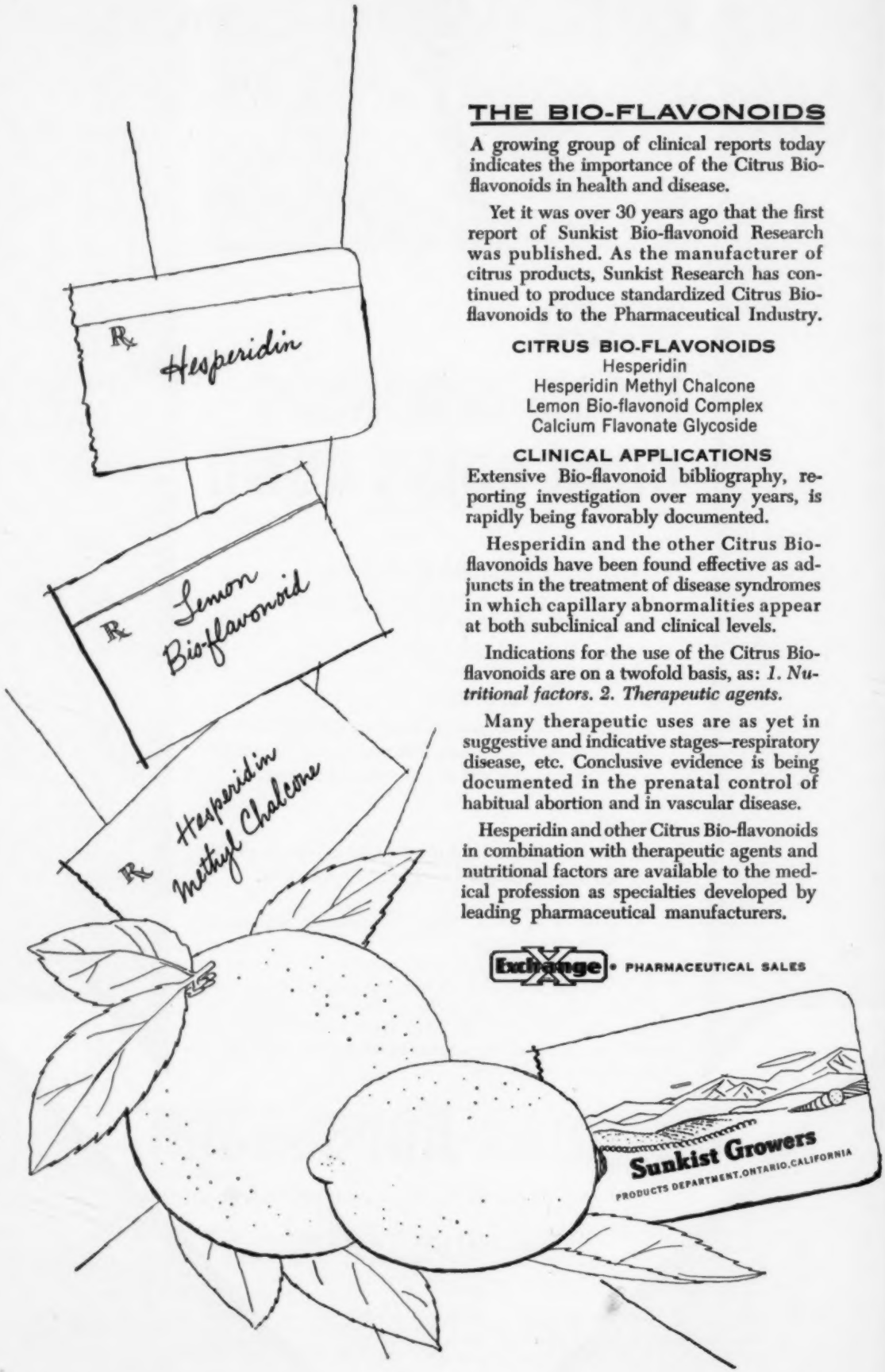


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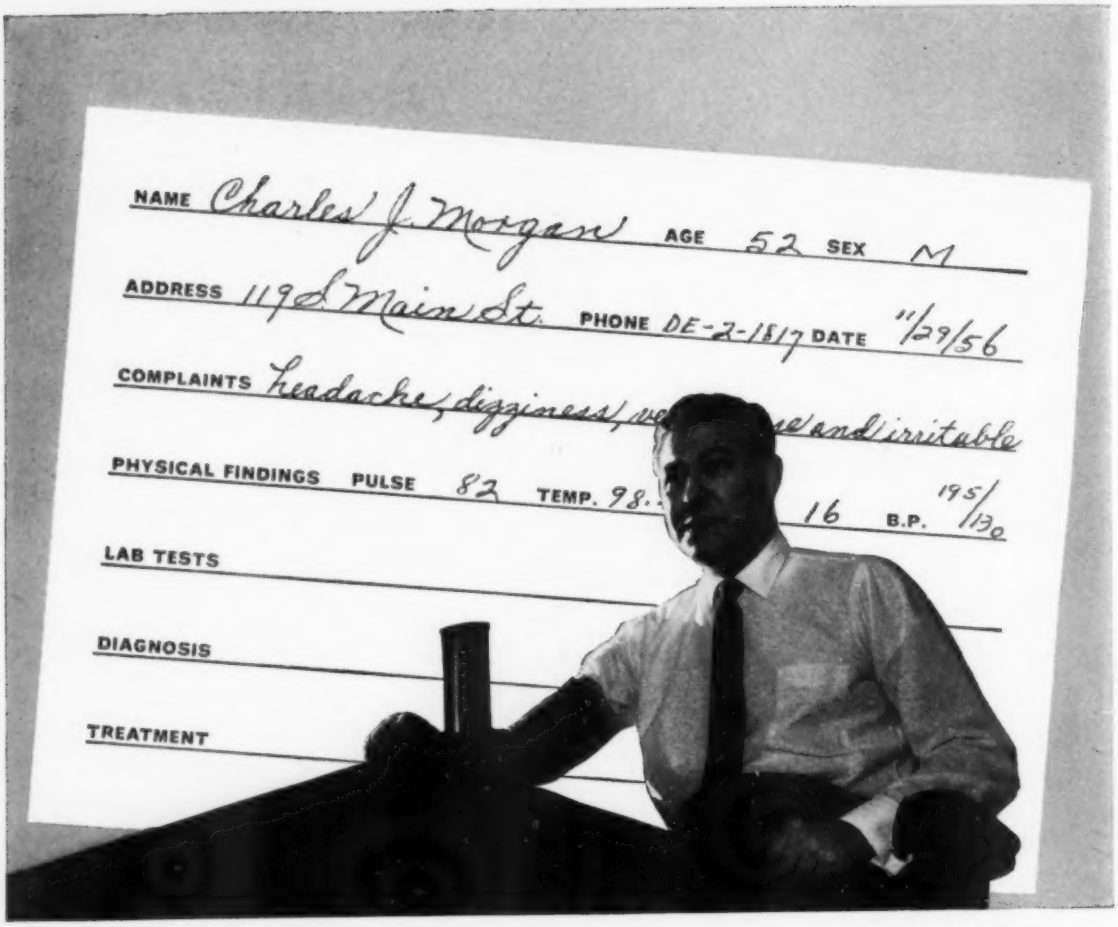
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1. LaBarbera, J.F.: Med.Rec. & Ann. *50*:242, 1956. 2. Ledbetter, P.V., and Morrow, E.J.: J. Am. Geriatrics Soc. *3*:172 (March) 1955. 3. Wilkins, R.W.: Am. J. Med. *17*:703 (Nov.) 1954. 4. Moyer, J.H.; Dennis, E., and Ford, R.: A.M.A. Arch. Int. Med. *96*:530, 1955.

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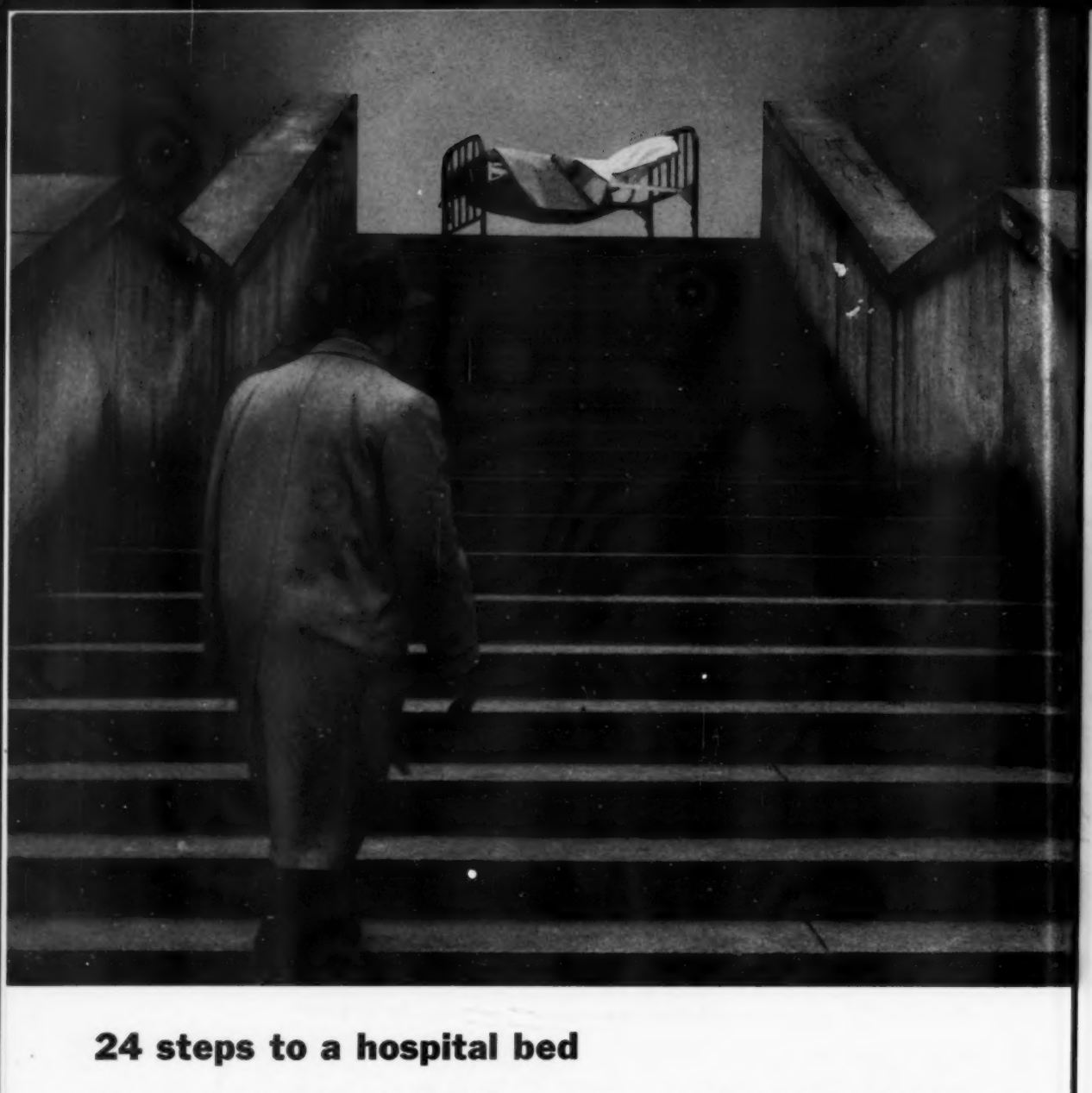

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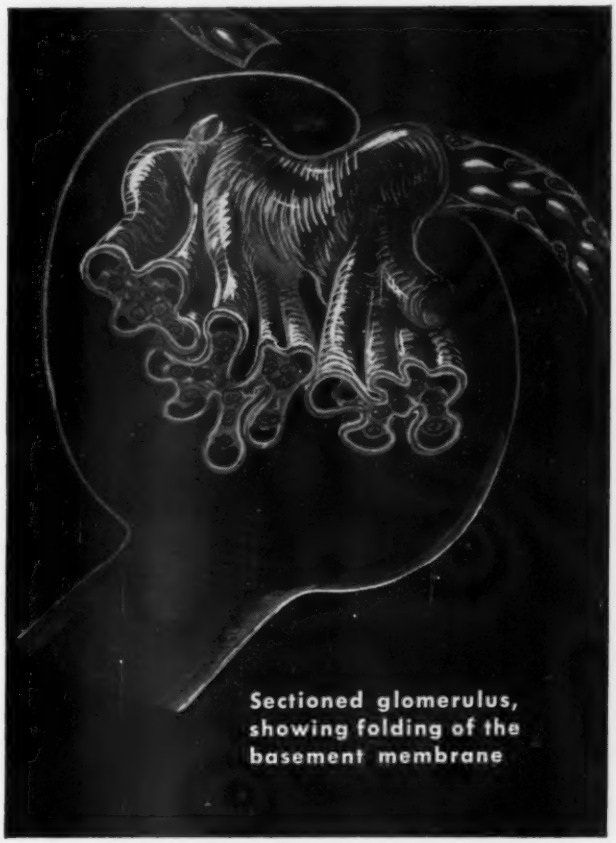

Sectioned glomerulus, showing folding of the basement membrane

*Settel, E.: Rolicton® (Aminoisometradine), a New, Nonmercurial Diuretic, Postgrad. Med. *21*:186 (Feb.) 1957.

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(1) Griffith, J. Q., Jr., *et al.*: Rutin and Related Flavonoids, Mack Publ. Co., Easton, Pa., 1955. (2) Griffith, J. Q., Jr.: Med. Clin. North America, November, 1954, p. 1717.

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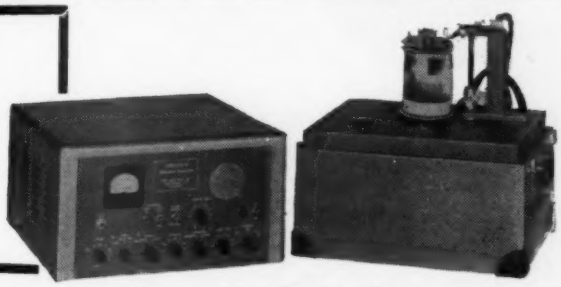

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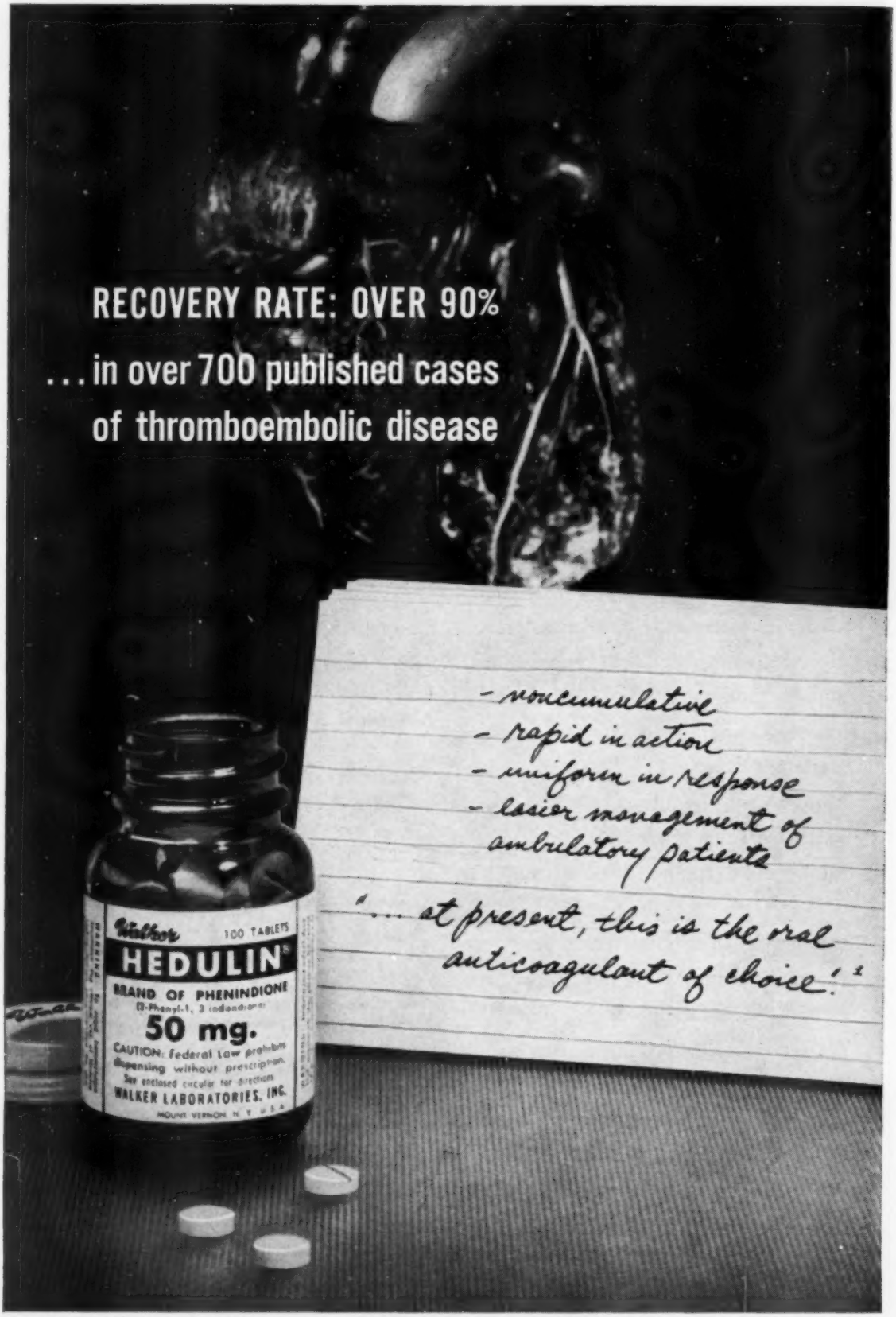
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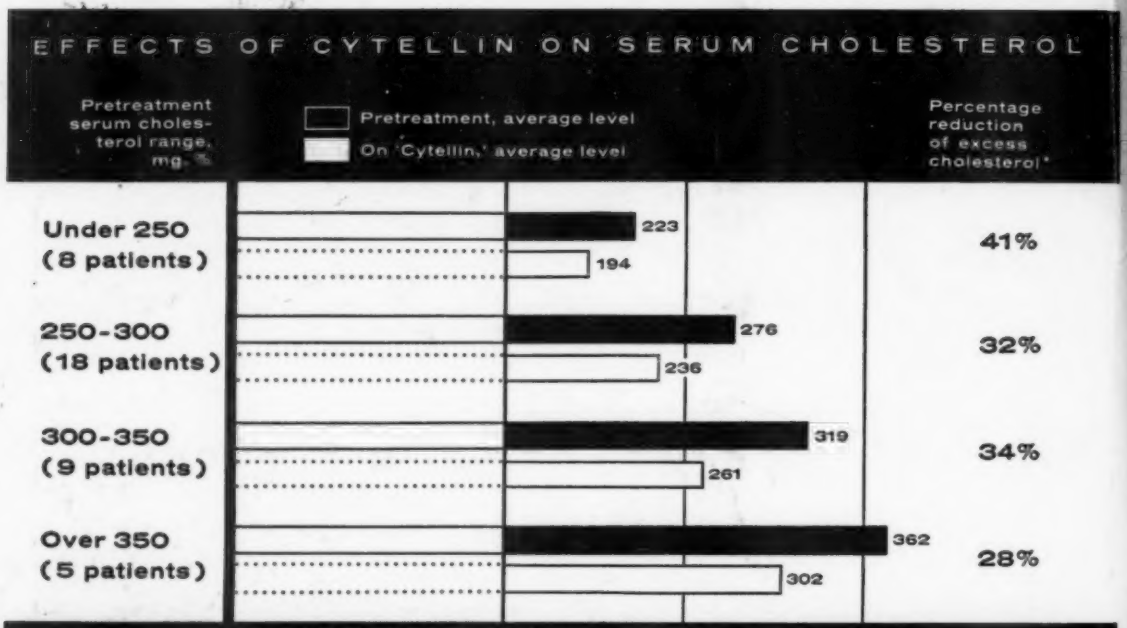
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194
41%
250-300 (18 patients)
276
236
32%
300-350 (9 patients)
319
261
34%
Over 350 (5 patients)
362
302
28%
Serum cholesterol, mg. % 150* 250 350
(Adapted from Am. J. Med. 17:61, 1955; Ohio Michigan M. Bull. 29:13, 1956; Am. J. M. Sciences 235:1, 1958; and Circulation, 14:77, 1956.)
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